Calcasieu Estuary Remedial Investigation/Feasibility Study (RI/FS): Baseline Ecological Risk Assessment (BERA)

Appendix I1: Assessment of Risks to Omnivorous Mammals in the Calcasieu Estuary

Prepared For:

CDM Federal Programs Corporation
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Under Contract To:

Mr. John Meyer, Regional Project Manager
U.S. Environmental Protection Agency, Region 6
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Prepared – October 2002 – By:

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Under Contract To:

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Appendix I1. Assessment of Risks to Omnivorous Mammals in the Calcasieu Estuary

1.0 Introduction

Development and industrialization in and around the Calcasieu estuary in southwestern Louisiana in recent decades has led to concerns of environmental contamination in the area. A Remedial Investigation/Feasibility Study (RI/FS) was commissioned to determine the risks posed by environmental contamination to ecological receptors inhabiting key areas of the Calcasieu Estuary. A Baseline Ecological Risk Assessment (BERA) is required to meet this objective. This appendix is part of the BERA and is conducted in accordance with the procedures laid out by the USEPA in the *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment* (USEPA 1997a). Under the eight-step process described by the USEPA for conducting a BERA, a screening ecological risk assessment (SERA) must first be conducted to determine preliminary estimates of exposure and risk.

The SERA for the Calcasieu Estuary (CDM 1999) identified areas of concern (AOCs), contaminants of concern (COCs), and ecological receptors potentially at risk. The SERA findings were revisited in a Baseline Problem Formulation (BPF) (MacDonald *et al*. 2001) to yield a refined list of contaminants of concern, areas of concern, and ecological receptors to be considered in the BERA. The Phase II data collection provided more information and, therefore, a better tool to estimate risk at a screening level. Using this information, a conservative, deterministic assessment was conducted and can be found in Appendix G, along with a description of the methods used to identify the COCs and AOCs for omnivorous mammals.

This appendix is organized as follows. Section 1 provides a brief overview of the results of the conservative, deterministic ERA for wildlife described in detail in Appendix G. The AOCs and COCs that screened through the conservative, deterministic assessment for omnivorous mammals are described in this section. Section 1 also includes a description of the conceptual model for omnivorous mammals in the Calcasieu Estuary. A statement outlining the purpose of this assessment concludes Section 1.

Section 2 describes the probabilistic risk assessment methods used to estimate risks of COCs to omnivorous mammals in the Calcasieu AOCs. Section 3 describes the probabilistic risk assessment results and Section 4 identifies the sources of uncertainty that could influence the estimated risks for omnivorous mammals. The final section of this appendix, Section 5, contains the conclusions regarding risks of COCs to omnivorous mammals in the Calcasieu Estuary.

1.1 Deterministic ERA Summary

The methods and results of the deterministic ecological risk assessment are presented in detail in Appendix G. In summary, the deterministic assessment used a conservative approach to estimate risk to omnivorous mammals from chemicals of potential concern (COPCs) in the Bayou d'Inde, Upper Calcasieu River, and Middle Calcasieu River AOCs. Several reference sites, including Bayou Connine Bois and Choupique Bayou, were also included in the deterministic assessment to provide a basis of comparison of risks. The deterministic assessment compared potentially attainable high exposures with conservative adverse effects benchmarks to provide a means of identifying which contaminants are a concern to omnivorous mammals and in which areas of the Calcasieu Estuary system. A risk quotient (total daily

intake/effect dose) approach was used to determine if the COPC screened through to the probabilistic ecological risk assessment, using the following decision rules:

- If all RQs were less than 1.0 for all areas of concern for a COPC, the COPC was eliminated from further consideration;
- If RQs were $\geq$1.0 for at least one area of concern, but were less than 1.2 times the RQs for references areas, the COPC was eliminated from further consideration. In these cases, the COPC is unlikely to be causing significant incremental risk in the area of concern over what is occurring in the background; and,
- If RQs were $\geq$1.0 for at least one area of concern and were $\geq$1.2 times the RQ of the reference area, the COPC screened through to the next phase.

COPCs that were screened through by the SERA are now refered to as contaminants of concern (COCs). Selenium was identified as a COC in all three areas, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and equivalents (TEQs) were identified in Middle Calcasieu River and Bayou d'Inde AOCs, and total PCBs and mercury were identified only in Bayou d'Inde AOC. The reference areas were also screened through to the probabilistic risk assessment so that risks in the AOCs could be compared to background risks. Results of the deterministic risk assessment are presented in Table I1-1.

1.2 Contaminants of Concern

The COCs that screened through to the probabilistic risk assessment for omnivorous mammals included mercury, TCDD-TEQs, total PCBs, and selenium and are described below.

Mercury

Mercury is found in the environment as the metal, Hg^0, and as divalent mercuric Hg(II) species. In the water column, Hg^0 is oxidized to Hg(II) under acidic conditions. Hg(II) undergoes a number of important reactions, one of which is methylation by microbes and adsorption and absorption by biota (Stein *et al*. 1996). Biomethylation occurs both in the sediments, where sulfate-reducing bacteria are the primary methylators of mercury, and in the water column (Winfrey and Rudd 1990). Methylation in the water column also occurs abiotically, mediated by dissolved organic carbon (Weber 1993). Methylmercury may make up as much as 25 percent of the mercury in rivers and lakes (Gilmour and Henry 1991).

Methylmercury is highly soluble in water, extremely mobile, and thus readily enters the aquatic food web. Because methylation is higher under anaerobic conditions, benthic organisms in the anaerobic zones of sediment may be exposed to high methylmercury concentrations. These organisms are consumed by a variety of species, including omnivorous mammals, leading to biomagnification up the food chain. The accumulation of methylmercury in aquatic organisms has been well documented, with concentrations in carnivorous fish 10,000 to >1,000,000 times the concentrations found in ambient waters (Stein *et al*. 1996). Gilmour and Henry (1991) showed that fish from contaminated systems may continue to contain high levels of methylmercury long after inputs to the systems have ceased. Also, the efficient assimilation of the lipophilic methylmercury in fat and muscle and lack of elimination results in increasing methylmercury concentrations with the age and size of fish and wildlife predators.

This assessment focuses on the risks posed by methylmercury to omnivorous mammals because this species of mercury is more readily bioaccumulated and more toxic to wildlife than is metallic mercury. Further, previous assessments of

methylmercury risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (MacIntosh *et al*. 1994; USEPA 1997b; Moore *et al.* 1999). Omnivorous mammals are moderately high in the food chain and are potentially at high risk of exposure to methylmercury because they consume invertebrates, fish, and waterfowl eggs (Environment Canada 2000; NSRL 2002) that are found in the Calcasieu Estuary system.

TCDD-TEQs

TCDD and TEQs represent a group of aromatic compounds with similar properties (WHO 1989). These terms refer to a specific group of polychlorinated dibenzo-*p*-dioxin (PCDDs) congeners, polychlorinated dibenzofuran (PCDFs) congeners and polychlorinated biphenyl (PCB) congeners. This group has a common structural relationship that includes lateral halogenation and the ability to assume a planar conformation. The planar conformation is important as it leads to a common mechanism of action in many animal species that involves binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah receptor- mediated biochemical and toxic response (van den Berg *et al.* 1998; Newsted *et al.* 1995; Safe 1994).

Each of these compounds, while similar in structure and acting at the same receptor, has different potencies, depending on the individual congener. To address these issues and effectively estimate the relative toxicity of these mixtures, a system has been created involving the development and use of toxic equivalency factors (TEFs). This approach is based on the *in vivo* and *in vitro* toxicity of each of the compounds in relation to TCDD. TCDD is considered to be the most toxic member of this class of chemicals (van den Berg *et al.* 1998; Birnbaum and DeVito 1995; Safe 1994) and the toxicity of the others depends on the degree of chlorination, the chlorination sites,

and the ability to achieve a planar form, relative to TCDD. There are a number of assumptions made when using the TEF approach. These include:

- The congeners are Ah-receptor antagonists and their toxicological potencies are mediated by their binding affinities; and,
- No interaction occurs between the congeners, and the sum of the individual congener effects accounts for the potency of the mixture.

The overall effect of these assumptions is a potency estimate or toxic equivalent (TEQ) value. A more detailed discussion of the TEF approach for expressing the toxicity of this class of chemicals is presented in Appendix G.

The environmental degradation and metabolism of the congeners varies due to their unique physical/chemical properties. These can cause substantial differences between the congeners detected in environmental samples and the congener makeup of the original product (van den Berg *et al.* 1998). The majority of the congeners have low solubility, low vapour pressure and high resistance to chemical breakdown, and are, therefore, highly persistent in the environment. They are also highly lipophilic with a high propensity to bind to organic and particulate matter. When released to aquatic systems, the majority of these compounds form associations with dissolved and/or particulate matter in the water column; biodegradation is considered to be a relatively minor fate process in water (NRCC 1981; Howard *et al*. 1991). Aquatic sediments provide a sink for these compounds and may represent long term sources to the aquatic food web (Kuehl *et al*. 1987; Muir *et al*. 1988; Corbet *et al*. 1983; Tsushimoto *et al*. 1982). As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Organisms may be exposed to TCDD-TEQs through trophic transfer. PCDDs, PCDFs and PCB congeners are highly bioaccumulative substances that increase in concentration as they are passed up the food chain (i.e., biomagnification). For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al*. (1994) observed that PCB concentrations increased from 935 ：g/kg in sediments, to 1,360 ：g/kg in bivalves, to 7,240 ：g/kg in oligochaetes, and to 64,900 ：g/kg in predatory gar pike. Mink are particularly sensitive to PCBs and similar chemicals (Moore *et al*. 1999). Research has found that they accumulate PCBs in their subcutaneous fat at levels 38 to 200 times dietary concentrations, depending on the PCB congener (USEPA 1993). The mammalian predators of the Calcasieu estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment estimates the risks posed by coplanar congeners to omnivorous mammals because these compounds are expected to biomagnify up the food chain. Further, previous assessments have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (Moore *et al*. 1999; Tillitt *et al*. 1996; Heaton *et al*. 1995). Omnivorous mammals are moderately high in the food chain and are potentially at high risk of exposure to coplanar congeners because they consume invertebrates, fish, and waterfowl eggs (Environment Canada 2000; NSRL 2002) found in the Calcasieu Estuary system.

Selenium

The fate of selenium and its compounds in the environment is influenced to a large degree by its oxidation state. The valence states of selenium range from -2 (hydrogen selenide) through 0 (elemental selenium), +2 (selenium dioxide), +4 (selenite) and +6 (selenate). The behavior of various compounds of selenium in the environment also

depends on ambient conditions including pH, the presence of metal oxides and biological activity (ATSDR 1996; Maier *et al*. 1988).

Elemental selenium is essentially insoluble and will remain inert when released in the environment under anaerobic conditions. Heavy metal selenides and selenium sulfides predominate in acidic soils and soils with high organic matter, and will remain insoluble and immobile in this form (NAS 1976). Selenites and selenates are water soluble and are, therefore, more bioavailable in surface water and water contained in soils (Eisler 2000a; ATSDR 1996; Robberecht and Van Grieken 1982). In general, these mobile forms of selenium dominate under aerobic and alkaline conditions. Sodium selenate is one of the most mobile selenium compounds in the environment because of its high water solubility and inability to adsorb onto particulates (NAS 1976). Selenium bioconcentrates and biomagnifies in aquatic food chains from invertebrates to birds (Ohlendorf *et al*. 1986a; 1986b; Lemly 1985; Saiki and Lowe 1987; Saiki *et al*. 1993). Lemly (1985) reported BCFs of 1,500-1,850 and BAFs of 1,746-3,975 for selenium in freshwater species. Concentrations of selenium in river otter and raccoon have been measured (wet weight) in various organs and ranged from 0.2 to 2.8 mg Se/kg (Wren 1984). These studies demonstrate that selenium has the potential to biomagnify up the food chain and accumulate in omnivorous mammals.

This assessment focuses on the risks posed by selenium to omnivorous mammals because this substance is expected to biomagnify up the food chain. Selenium bioconcentrates in aquatic food chains from invertebrates to birds with diet being identified as the primary source for fish and piscivorous birds having the highest body burdens among avians (Eisler 2000a). Omnivorous mammals are fairly high in the food chain and are potentially at high risk of exposure to selenium because they

consume invertebrates, fish, and waterfowl eggs (Environment Canada 2000; NSRL 2002) that are found in the Calcasieu Estuary system.

Total PCBs

Polychlorinated biphenyls (PCBs) is the generic term applied to a group of 209 chlorinated organic compounds that have similar molecular structures and properties. The majority of PCB congeners have low solubility, low vapor pressure and high resistance to chemical breakdown. Due to chemical stability, PCBs are highly persistent in the environment.

PCBs are persistent and highly lipophilic substances with low water solubility and a high propensity to bind to organic and particulate matter. In bulk releases to aquatic compartments, these substances will tend to remain as a non aqueous phase liquid and settle to the bottom of the water body. Here, PCBs gradually adsorb to organic and particulate matter and remain sequestered in sediment layers. Exposure to PCBs from this compartment occurs as a result of benthic organisms ingesting sediments during foraging and when sediments are stirred and PCB-laden particles resuspended in the water column. As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Predatory organisms may be exposed to chemical contaminants through trophic transfer. Organisms lower in the food chain may ingest and accumulate a substance, which is then passed on when they are consumed by higher food chain predators. Benthic communities are at the highest risk of direct exposure to PCBs. Benthic invertebrates will be exposed to PCBs through direct contact with interstitial pore water, ingestion of sediment particles, and ingestion of organisms that have also been exposed to contaminants. Pelagic organisms in the Calcasieu estuary will be exposed to PCBs through dermal and gill contact with surface waters; ingestion of water,

suspended sediment, and organic matter; ingestion of sediment for bottom-feeding fish; and ingestion of other benthic and pelagic organisms. Uptake of PCBs by fish occurs mainly through the gills and the gastrointestinal tract (Shaw and Connell 1984). Most PCB accumulation in top fish predators can be attributed to the food pathway (Thomann 1989). Other species, such as amphibians, are also exposed to PCB-contaminated surface waters. Insectivorous, carnivorous, and piscivorous birds and mammals that reside, or partially reside, within the estuary are exposed to PCBs principally through diet and trophic transfer. PCBs are highly bioaccumulative substances that increase in concentration as they are passed up the food chain (Eisler and Belisle 1996). For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al*. (1994) found that PCB concentrations increased from 0.935 mg/kg in sediments, to 1.36 mg/kg in bivalves, to 7.24 mg/kg in oligochaetes, and to 64.9 mg/kg in predatory gar pike. PCBs have also been observed to biomagnify in several birds (Bosveld and van den Berg 1994; Eisler 2000b). The avian and mammalian predators of the Calcasieu estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment focuses on the risks posed by total PCBs to omnivorous mammals because PCBs are expected to biomagnify up the food chain (Eisler and Belisle 1996; Hoffman *et al*. 1996). Further, previous assessments of PCBs risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (Moore *et al*. 1999). Omnivorous mammals are fairly high in the food chain and are potentially at high risk of exposure to PCBs because they consume invertebrates and fish found in the Calcasieu Estuary.

1.3 Receptors of Concern

Thorough observations of the study area led to the identification of several mammalian species including bats (Order *Chiroptera*), rabbit (*Sylvilagus* sp.), raccoon (*Procyon lotor*), muskrat (*Ondatra zibethicu*s), marsh rice rat (*Oryzomys palustri*s), eastern fox squirrel (*Sciurus niger*), nutria (*Myocastor coypus*), river otter (*Lutra canadensis*), white-tailed deer (*Odocoileus virginianus*), and dolphins (*Delphinidae*; ChemRisk 1996). The number of omnivorous mammalian species that feed on aquatic prey and have the potential to occur in the study area is quite limited.

The exposure assessment for omnivorous mammals will be based on a hypothetical receptor that incorporates the characteristics typical of this receptor group. This hypothetical receptor is based upon the characteristics of three aquatic-dependent omnivorous mammals commonly found in the Calcasieu Estuary: raccoons, muskrats, and marsh rice rats. These omnivores consume aquatic invertebrates and fish as parts of their diets. The following sections review the life histories and foraging behaviors of these three species. This information is then used to develop the life history and foraging behavior of the hypothetical receptor that will be used to represent omnivorous mammals in this assessment.

Raccoon (*Procyon lotor*)

Raccoons are stocky, short-legged, grayish to blackish animals (with some yellow and white) that are about the size of a large beagle. Raccoons are the most abundant and widespread omnivore in North America, and are common in Louisiana (USEPA 1993). Adult males and females range in length from 46 to 71 cm with a 20 to 30 cm tail. Their weight can range from 3 to 7.5 kg for females and up to 9 kg for males (USEPA 1993). The young grow rapidly and are soon indistinguishable from adults. Raccoons produce only one litter per year. Mating occurs from December to June.

The young are generally born in late April or early May after a gestation period of 63 days. The number of young in a litter is usually two to five.

Raccoons occur in southern Canada, most of the United States with the exception of the Rocky Mountains and desert areas of the southwest, and in Mexico and most of Central America (Lowery 1974; Choate *et al.* 1994). The raccoon is usually most abundant near water, especially in bottomland forests along streams, hardwood swamps, flooded timber near reservoirs, marshes, wooded areas near urban developments, and agricultural areas. They are able to live in a diversity of habitats provided that certain requirements are found nearby, such as food, water, and a protected area for denning (USEPA 1993). The size of a raccoon's home range can vary dramatically depending on the availability of food and shelter, as well as the space necessary for reproduction. In riparian areas of Michigan, home ranges between 0.2 and 8 km^2 have been reported (USEPA 1993), however, depending on habitat and food availability, the size of the range may be anywhere from 0.1 km^2 up to 50 km^2 (Environment Canada 2000).

Raccoons eat crayfish, snails, clams, small fishes, frogs, earthworms, bird and turtle eggs, and a wide variety of insects such as grasshoppers, crickets, and beetles. Acorns, berries, watermelons, cantaloupe, corn, tomatoes, and the tender shoots and buds of many trees and other plants are also consumed. (Lowery 1974; Choate *et al.* 1994). In the spring and early summer, raccoons will eat more animal than plant material, with fruit being the primary nutrition source in the summer (USEPA 1993).

Marsh rice rat *(Oryzomys palustri*s)

This species is a small rat with a scantily haired tail equal in length to the head and body combined. The upper parts are dark gray, mixed with black, and the underparts are grayish white. The sexes are alike in color and size, with weights ranging from

30 - 78 g (enature 2002). Rice rats are highly fecund and can produce as many as seven litters per year. However, because the life span of a female is less than one year, the average number of litters is probably no more than five or six (Lowery 1974; Choate *et al.* 1994).

The marsh rice rat ranges throughout the state of Louisiana and is more abundant in the southern half of the state. It occurs in the southeastern United States and most of Central America. The marsh rice rat's habitat is always rather wet, marshy places such as canal banks, grassy ditches, the edges of swamps, lakes, ponds, bayous, and streams, and fields with wet soil. The marsh rice rat can be found in marshes not subject to high tides and flooding. It is a good swimmer and diver, and is seldom, if ever, found in dry fields or well-drained woodlands far from water.

Marsh rice rats eat mostly seeds and the succulent parts of various available plants, but also eat carrion, insects, snails, crustaceans, clams, bird eggs, and baby birds and turtles (Lowery 1974; Choate *et al.* 1994; Rose and Dueser 1999).

Muskrat *(Ondatra zibethicus*)

Muskrats are fairly large rodents with head and body measuring 25 to 36 cm and with a 20 to 25 cm tail (Burt and Grossenheider 1980). The rear feet are webbed with fringes of stiff hairs, and the scaly tail is vertically flattened. The upper fur is dense with guard hairs and is a rich dark brown and the underparts are gray to white (Lowery 1974; Choate *et al.* 1994). Adult weights can range from 0.5 kg to over 2 kg, and males are slightly larger than females (Dozier 1950; Parker and Maxwell 1984). They reproduce year round. The adult female is capable of producing seven to eight litters per year.

Muskrats occur in Canada, Alaska, and most of the continental United States. In Louisiana, they occur throughout the state and are especially numerous in the coastal marshes. They range inland where rice is grown and are found along bayous and lakes throughout most of southern Louisiana. Muskrats live in saline, brackish, and freshwater marshes, ponds, sloughs, lakes, ditches, streams, and rivers (Lowery 1974; Choate *et al.* 1994).

Muskrats consume mainly aquatic vegetation but also a variety of animals (Dozier 1953; Errington 1939)**.** The muskrat's diet is comprised chiefly of the roots and basal portions of aquatic plants, but also include the shoots, bulbs, tubers, stems, and leaves (Dozier 1950; 1953; Willner *et al*. 1980; Svihla and Svihla 1931). Marsh grasses, cattails, and sedges (Svihla and Svihla 1931; Johnson 1925; Willner *et al*. 1975; Kiviat 1978) also seem to be important in the muskrat's diet. Animals that muskrats consume include turtles, crabs, crayfish, mussels, frogs, young birds and small fish (Lowery 1974; Choate *et al.* 1994; Errington 1939; Johnson 1925; Willner *et al*. 1980).

Hypothetical Omnivorous Receptor of Concern

The hypothetical omnivorous mammal for this assessment is based upon the behavior and characteristics described for raccoons, muskrats, marsh rice rats, and other omnivorous mammals inhabiting the area.

- The receptor body weight is assumed to equal the average of the species with a coefficient of variation equal to 13%. This value is an average of coefficients of variation obtained from various body weight studies of raccoons and muskrats. A "what if" scenario will also be conducted to estimate the risk to a small omnivorous mammal. Small mammals

generally have elevated exposures because they have higher food intake rates (when normalized to body weight) than do larger mammals.

- The hypothetical receptor is assumed to have a relatively small foraging range with high site fidelity and no territoriality. Bayou d'Inde, Middle Calcasieu River, and Upper Calcasieu River AOCs were identified as areas of concern for omnivorous mammals in the deterministic risk assessment. It is assumed that receptors will forage exclusively within each of these areas.
- The diet is assumed to consist of equal parts animal and vegetable matter. The animal portion is assumed to be equally divided among fish, aquatic invertebrates and terrestrial invertebrates.
- Omnivorous mammals in the area are opportunistic in terms of habitat as well as diet. For this assessment, we assume that hypothetical omnivore receptors will be found in most habitats in the AOCs.
- The hypothetical receptor is assumed to be resident year-round in the Calcasieu area. The temporal scale for this assessment is long term because: (1) contaminant levels are unlikely to exhibit large temporal variability because of their high persistence, and (2) chronic toxicity typically occurs at much lower levels than acute toxicity.

1.4 Conceptual Model

The conceptual model illustrates the relationships between sources and releases of COCs, their fate and transport, and the pathways through which COCs reach omnivorous mammals and exert potential adverse effects. The model enhances the level of understanding regarding the relationships between human activities and ecological receptors at the site under consideration. In so doing, the conceptual

model provides a framework for predicting effects on ecological receptors and a template for generating risk questions and testable hypotheses (USEPA 1997a; 1998). The conceptual site model developed for the Calcasieu Estuary is described in greater detail in Chapter 7 of the BPF. It summarizes information on the sources and releases of COCs, the fate and transport of these substances, the pathways by which ecological receptors are exposed to the COCs , and the potential effects of these substances on the ecological receptors that occur in the Calcasieu Estuary. In turn, this information is used to develop a series of risk hypotheses that provide predictions regarding how ecological receptors will be exposed to and respond to the COCs.

Omnivorous mammals are exposed to a number of COPCs in the Calcasieu Estuary system and the deterministic risk assessment (Appendix G) identified those COCs that pose potential risks to these animals. Specifically, omnivorous mammals are at greatest risk from mercury, selenium, total PCBs, and TCDD-TEQs in the Calcasieu Estuary. These substances are persistent and bioaccumulative and are available for uptake by omnivorous mammals, primarily through the food chain. The Phase II sampling program provided data identifying substantial tissue residues of these substances in fish and aquatic invertebrates, which are prey items of many omnivorous mammals. Other routes of exposure, including inhalation, water consumption and sediment ingestion have been excluded from this assessment as their contribution to overall exposure is likely negligible (Moore *et al*. 1997; 1999).

1.5 Assessment Endpoints

An assessment endpoint is an 'explicit expression of the environmental value that is to be protected' (USEPA 1997a). The selection of assessment endpoints is an essential element of the overall ERA process because it focuses assessment activities

on the key environmental values (e.g., reproduction of omnivorous mammals) that could be adversely affected by exposure to environmental contaminants. Assessment endpoints must be selected based on the ecosystems, communities, and species that occur, have historically occurred, or could potentially occur at the site (USEPA 1997a).

To support the identification of key assessment and measurement endpoints for the Calcasieu Estuary BERA, the United States Environmental Protection Agency (USEPA) convened a BERA workshop in Lake Charles, LA on September 6 and 7, 2000. The workshop participants included representatives of the USEPA, United States Geological Service (USGS), National Oceanic and Atmospheric Administration (NOAA), Louisiana Department of Environmental Quality (LDEQ), United States Fish and Wildlife Service (USFWS) and CDM Federal. The workshop was designed to enable participants to articulate the goals and objectives for the ecosystem (i.e., based on the input that had been provided by the community in a series of public meetings), to assess the state of the knowledge base, to define key issues and concerns, and to identify the chemicals and areas of potential concern in the study area. This workshop provided a basis for refining the candidate assessment endpoints that had been proposed based on the results of the SERA (CDM 1999). Workshop participants also identified a suite of measurement endpoints that would provide the information needed for evaluating the status of the assessment endpoints (MacDonald *et al*. 2000a).

Aquatic-dependent mammals are integrally linked to aquatic ecosystems as a result of their reliance on aquatic organisms for food. These species can be classified based on their feeding habits into two main groups: omnivorous mammals (i.e., species that eat a wide variety of plants and animals, including aquatic organisms) and piscivorus mammals (i.e., species that eat primarily fish). Due to their reliance on aquatic

organisms for food, it is important to evaluate the effects of environmental contaminants on omnivorous mammals.

Although mammals can be exposed to environmental contaminants through dermal contact with contaminated surface water or sediments (i.e., dermal exposure), inhalation of volatilized contaminants, or consumption of contaminated surface water, the bulk of their exposure is associated with the consumption of contaminated prey items. This is especially true for persistent and bioaccumulative COCs. Therefore the assessment endpoint for the assessment of risks to omnivorous mammals in the Calcasieu Estuary is the survival, growth, and reproduction of omnivorous mammals.

1.6 Measurement Endpoints

A measurement endpoint is defined as 'a measurable ecological characteristic that is related to the valued characteristic selected as the assessment endpoint' and it is a measure of biological effects (e.g., mortality, reproduction, growth; USEPA 1997a). Measurement endpoints are frequently numerical expressions of observations (e.g., toxicity test results, community diversity measures) that may or may not be compared to similar observations at a control and/or reference site.

A single measurement endpoint will be used to evaluate the risks to aquatic-dependent omnivorous mammals. The potential for adverse effects on omnivorous mammals, such as the raccoon, will be evaluated using prey tissue data. The following measurement endpoint applies to omnivorous mammals: the comparison of modeled daily contaminant intake, based on contaminant residues in prey tissues, to the results of laboratory toxicity studies.

Specifically, the data on the concentrations of contaminants measured in invertebrates and small fish (i.e., <15 cm in length) and aquatic invertebrates (< 12.5 cm in length) will be used. The prey tissue data will be compiled by geographic area within the estuary (based on the diet and foraging range of a hypothetical mammal species), incorporated into a daily intake exposure model, and compared to appropriate toxicity values for survival and reproduction of omnivorous mammals. In this evaluation, the tissue residue data for the fish and aquatic invertebrates collected in the estuary will be assumed to be similar to that for other species not captured during the sampling program. Tissue samples with concentrations of one or more COCs in excess of one or more toxicity thresholds will be considered to have contaminant concentrations sufficient to potentially adversely affect the survival or reproduction of omnivorous mammals. The concentrations of contaminants in fish tissues from Calcasieu Estuary AOCs will also be compared to the levels measured in fish collected from the reference areas to determine if the risks associated with the consumption of prey items from the Calcasieu Estuary AOCs are higher than those determined for reference sites.

1.7 Risk Hypothesis and Questions

The following risk hypothesis was developed to identify the key stressor-effect relationships that will be evaluated in the ecological risk assessment:

> Based on the physical-chemical properties (e.g., K_{ow}s) of the bioaccumulative contaminants of concern, the nature of the food web in the Calcasieu Estuary, and the effects that have been documented in laboratory studies, mercury, selenium, total PCBs, and TCDD-TEQs released into surface waters will

accumulate in the tissues of aquatic organisms to levels that adversely affect the survival, growth, and/or reproduction of omnivorous mammals.

To assess ecological risks, the assessment endpoint must be linked to the measurement endpoint by risk questions. In this study, the investigation to assess the risks of COCs to mammals was designed to answer the following risk questions:

- Are the levels of contaminants in the tissues of prey species of omnivorous mammals in the Calcasieu Estuary higher than the tissue residue benchmark values for survival, growth, or reproduction?
- If yes, what are the probabilities of effects of differing magnitude for survival, growth, or reproduction of omnivorous mammals?

The linkages between the assessment endpoint and the measurement endpoints are articulated in greater detail in Table A1-21 of the Baseline Problem Formulation (MacDonald *et al*. 2001).

1.8 Purpose of Appendix

The purpose of this assessment is to determine the veracity of the above risk hypothesis by characterizing the risks posed to the omnivorous mammalian community associated with exposure to the COCs identified in Appendix G.

2.0 Methods

A step-wise approach was used to conduct a probabilistic assessment of risks to the omnivorous mammalian community posed by the COCs in the Calcasieu Estuary. The three main steps in this process included:

- Collection, evaluation, and compilation of the relevant information on the concentrations of COCs in prey items in the Calcasieu Estuary (2.1);
- Implementation of a deterministic assessment of risks to omnivorous mammals, including the identification of contaminants of concern (COCs) and areas of concern (AOCs; Appendix G); and, for those COCs that screened through;
- Assessment of the exposure of omnivorous mammals to COCs (Figure I1-1);
- Assessment of the effects of COCs on omnivorous mammals (Figure I1-2); and,
- Characterization of risks to the omnivorous mammalian community (Figure I1-3).

Each of these steps is described in this Appendix. The results of the deterministic assessment were briefly reviewed in Section 1.1. For details of this assessment, see Appendix G.

2.1 Collection, Evaluation, and Compilation of Data

Information on chemical levels in tissues of prey of omnivorous mammals were collected in two phases, termed the Phase I and Phase II sampling programs. The

Phase I program results indicated that the detection limits for many of the COCs in tissues were orders of magnitude above corresponding benchmarks. Therefore, the Phase I results for tissues were not considered in this assessment. The methods used to collect the tissue samples, quantify the levels of COCs, evaluate the reliability of the data, and compile the information in a form that would support the BERA are described in the following sections.

Sample Collection of Tissues - More than 600 tissue samples were collected at sites located throughout the estuary between October, 2000 and November, 2000.

Biota tissue samples were collected in three AOCs in the estuary (Upper and Middle Calcasieu River, and Bayou d'Inde) and in the reference areas (Bayou Choupique, Grand Bayou, Bayou Bois Connine, Johnson Bayou, Willow Bayou). There were also a number of sub-areas within the AOCs from which samples were taken. The USEPA Region V FIELDS tools were used to randomly select coordinates (i.e., latitude and longitude) for the assigned number of primary sampling stations and alternate sampling stations (i.e., which were sampled when it was not possible to obtain samples from the primary sampling stations). In the field, each sampling station was located with the aid of navigation charts and a Trimble differentially-corrected global positioning system (GPS). Using standard statistical power analysis methods, an evaluation of previously collected data was completed to determine the number of samples to be collected within each area and sub-area.

The methods used to collect, handle, and transport the tissue samples are described in CDM (2000a; 2000b; 2000c; 2000d; and 2000e). Briefly, fish and invertebrate species were collected by hook and line, hand collection and netting. Minnows and other small bait species were collected using legal cast nets, minnow traps, dip nets and bait seines in accordance with the Louisiana Department of Wildlife and

Fisheries. Each sample was wrapped in aluminum and put in a Ziploc® bag. All samples were kept frozen and shipped to laboratories in coolers on dry ice.

Chemical Analyses of Tissues - Chemical analysis of the tissue samples was conducted at various contract laboratory program (CLP) and subcontract (non-CLP) analytical laboratories, including USEPA Region VI Laboratory, USEPA Region VI CLP laboratories, Olin Contract laboratories, Texas A&M University laboratories, ALTA laboratories, AATS laboratories and EnChem laboratories. Upon receipt at the laboratory, tissue samples were held in freezers until analysis.

All tissue samples were analyzed for total target analyte list (TAL) metals, target compound list (TCL) semi-volatile organic compounds (SVOCs) and TCL pesticides. Total metals were quantified using the SW6010B method. Polycyclic aromatic hydrocarbons and/or other semi-volatile organic compounds were quantified using the SW8270C method. Methods SW8081A and SW8082 were used to quantify pesticides. Twenty percent of the tissue samples were analyzed for PCB congeners and dioxins/furans. EPA Method SW1668 was used to quantify PCB congeners and SW8290 was used for dioxins/furans.

EnChem laboratories used additional analytical methods to quantify mercury, polycyclic aromatic hydrocarbons (PAHs), pesticides and dioxins and furans. Methods 1631MOD and 1630MOD were used to quantify mercury and methylmercury, respectively. PAHs were quantified using Method 8270C-SIM. Method SW8082 and AXYS Method CL-T-1668A/Ver.3 were used to quantify pesticides. Dioxins and furans were quantified using AXYS Method DX-T-8290/Ver.2.

Data Validation and Verification - All of the data sets generated during the course of the study were critically reviewed to determine their applicability to the assessment of risks to the biotic community in the Calcasieu Estuary. The first step in this process involved validation of the tissue chemistry data. Following translation of these data into database format, the validated data were then further evaluated to ensure the quality of the data used in the risk assessment**.** We were unable to confirm tissue data results against the original source.

Database Development - To support the compilation and subsequent analysis of the information on biota in the Calcasieu Estuary, a relational project database was developed in MS Access format. All of the tissue chemistry data compiled in the database were georeferenced to facilitate mapping and spatial analysis using geographic information system (GIS)-based applications (i.e., ESRI's ArcView and Spatial Analyst programs). The database structure made it possible to retrieve data in several ways, including by data type (i.e., chemistry vs. toxicity), by stream reach (i.e., Upper Bayou d'Inde vs. Lower Bayou d'Inde), by sub-reach (i.e., Upper Bayou d'Inde-1 vs. Upper Bayou d'Inde-2), and by date (i.e., Phase I vs. Phase II). As such, the database facilitated a variety of data analyses.

2.2 Probabilistic Ecological Risk Assessment

Monte Carlo analysis is an increasingly widely used approach to probabilistic risk assessment (USEPA 1997c; 1999). It is used to propagate uncertainty associated with the variability of input variables, as well as any incertitude associated with how to parameterize input distributions. In this assessment we use probability bounds analysis to determine the relative contributions of incertitude and variability to

exposure estimates (see Chapter 9 of MacDonald *et al*. 2001 for more information on the uncertainty analysis approaches used here).

Monte Carlo analysis requires the specification of the statistical distributions of each of the input variables and their interdependencies as measured by correlations. Computer software such as Crystal Ball is used to 'sample' from these distributions and, via the exposure model equation, compute an exposure distribution. This process is repeated many times so as to build up a histogram that serves as the estimate of the full distribution of exposures (explicitly including the tail risks of extreme exposure).

Probability bounds analysis is an exact numerical approach (not based on simulation) that takes as input the same probability distributions used in Monte Carlo simulation, or, when they are difficult to specify precisely, bounds on these distributions (Ferson *et al*. 2002). The method then rigorously computes bounds on the cumulative distribution function. The spread between the bounds of an input or output distribution corresponds directly to the amount of incertitude we have about how to describe the variable. Probability bounds analysis is also useful when independence assumptions are untenable, or when sparse empirical data make it difficult to quantify the correlations among variables.

2.2.1 Exposure Characterization

We estimate exposure of omnivorous mammals to methylmercury, selenium, total PCBs, and TCDD-TEQs via a daily intake model that considers the dietary ingestion route of exposure. Omnivorous mammals are unlikely to use the saline waters of Bayou D'Inde as a source of drinking water and the inhalation route of exposure has

been shown to be an insignificant source of hydrophobic contaminants in previous assessments of the risks of these substances to aquatic-dependent wildlife (*e.g.*, Moore *et al*. 1999). Sediment ingestion was also considered as a possible route of exposure, however, deterministic analysis indicated that the contribution of sediment intake to overall exposure of COCs was insignificant. Therefore, the exposure model used in this assessment only includes the ingestion of food items as an exposure route. Tissue concentrations of methylmercury, selenium, total PCBs, and TCDD-TEQs in plants and terrestrial insects, important nutrient sources for omnivorous mammals, were not available for this exposure characterization, and so, total exposure to these substances may be underestimated. This exposure assessment assumes that the hypothetical receptor is present year round in each of the identified areas of concern.

The temporal scale for this assessment is long term because: (1) levels of mercury, selenium, total PCBs, and TCDD-TEQs are unlikely to exhibit high temporal variability, and (2) chronic toxicity occurs generally at lower levels than acute toxicity. The spatial scale of this assessment is considered to be consistent with home ranges reported for omnivorous mammals (USEPA 1993). The foraging area for the hypothetical receptor is set to 25,000 m^2. This area is equivalent to a circular zone of 180 metres in diameter or 1,000 metres of shoreline 25-m wide, both of which fit within the identified Areas of Concern.

The exposure model calculates the total daily intake of methylmercury, selenium, total PCBs, and TCDD-TEQs associated with the ingestion of food. Chemical assimilation efficiency terms are not included in the exposure equation because the efficiencies of chemical adsorption in wild animals following ingestion will likely be similar to the efficiencies in laboratory animals exposed to the substances in toxicity

studies. Thus, the chemical assimilation efficiency terms will cancel out when the exposure and effect estimates are combined to estimate risk.

The exposure model is adapted from USEPA (1993) and is represented in Equation 1:

$$TDI = FMR \times \sum_{i=1}^{n} \frac{C_i \times P_i}{AE_i \times GE_i} \quad \textbf{(1)}$$

where:

TDI = total daily intake of substance (mg/kg BW/day),

FMR = normalized free metabolic rate (Kcal/kg BW/day),

i = 1 = fish, 2 = invertebrates

C_i = concentration of substance in prey (mg/kg ww prey),

P_i = proportion of prey in the diet (unitless),

GE_i = gross energy of prey (Kcal/kg ww prey),

AE_i = assimilation efficiency of prey (unitless),

Each input variable is described in the following sections, including the parameterizations for the Monte Carlo analyses and the probability bounds analyses.

2.2.1.1 Selection of Criteria for Input Distributions

The distributions and distribution parameters used in the exposure analyses are summarized in Tables I1-2 (Monte Carlo parameters) and I1-3 (probability bounds parameters). Input distributions were assigned as follows: lognormal distributions for variables that are positively skewed with a lower bound of zero and no upper bound (e.g., tissue concentrations), beta distributions for variables bounded by zero and one (e.g., prey assimilation efficiency), and normal distributions for variables that

are symmetric and not bounded by one (e.g., body weight). The lognormal distribution is often used to provide good representations for physical quantities constrained to being non-negative, and that are positively skewed, such as contaminant concentrations, stream flows, or magnitudes of accidents (Small 1990). Ott (1995) provides an extensive discussion of the theoretical reasons for why contaminant concentrations in the environment are expected to be lognormally distributed. The beta distribution provides a flexible means of representing variability over a fixed range, such as zero to one (Small 1990). The beta distribution can take on a wide variety of shapes between the fixed endpoints and this flexibility has led to its empirical use in diverse applications. The normal distribution arises in many cases because of the central limit theorem, which results in a normal distribution for additive quantities such as body weights (Small 1990). The normal distribution can often be used for variables that are non negative, as long as coefficients of variation (CV) are small (e.g., body weight). This is because many distributions converge to a normal distribution as CVs become small. With most random number generators, it is impossible to obtain numbers more than five standard deviations from the mean. Thus, as long as the CV is less than 0.2, there is no concern for selecting negative values for non-negative variables.

2.2.1.2 Input Distributions

Body Weight

Although body weight data are not used in the exposure model directly, they are a required variable in allometric models used to estimate the free metabolic rate. For this assessment, we used body weights that represent an average-sized and a small-sized omnivorous mammal.

For the Monte Carlo analysis, the mean body weight of raccoons, muskrats, and marsh rice rats was used (2602 g). Because the feeding guild encompasses species with widely varying body weights, the calculation of the standard deviation of the mean body weight would have yielded an unduly wide distribution. Instead, we adopted a coefficient of variability (CV) of 13%, which is typical of the body weight distribution for many omnivorous mammal species (USEPA 1993). The application of the adopted CV yielded a standard deviation of 340.

We also repeated the Monte Carlo analysis with a mean body weight of 55.0 g (standard deviation equal to 7.2). This body weight is representative of the smallest mammals in the guild. Small mammals tend to have higher metabolic rates and, as a result, may be at higher risk of exposure.

Body weights were assumed to be distributed normally. The uncertainty in this variable is likely due to variability, with little incertitude. Thus, probability bounds were not established for this input variable.

Free metabolic rate (FMR)

Nagy (1987) derived an allometric equation for estimating the metabolic rate of free-living mammals using the general equation:

$$\text{FMR (kJ/d)} = a \ \text{BW (g)}^{b} \qquad (2)$$

For both the Monte Carlo and the probability bounds analyses, *FMR* for omnivorous mammals was estimated with a probabilistic approach wherein distributions were derived for each of the input variables [body weight (*BW)*, *a*, *b*] and combined according to Equation 2. The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy (1987), assuming an underlying normal distribution for each. For eutherian mammals, log *a* had a reported mean of 0.525 and a standard

error of 0.057, and *b* had a reported mean of 0.813 and a standard error of 0.023 (Nagy 1987). The body weight (*BW*) distribution was described above.

Proportion of Prey Items in the Diet

A number of studies have investigated the dietary composition of raccoons (Alexander 1977; Dorney 1954; Hamilton 1936; Hamilton 1940; Hamilton 1951; Llewellyn and Uhler 1952) based on the percent volume or weight of digestive tract items. Being omnivores, the diet of raccoons varies considerably, and animal matter comprises anywhere from 13 to 75% of the diet. Dietary studies of muskrat indicate that animal matter is usually absent from the diet, with only one investigation noting a 5% presence (O'Neil 1949). Muskrats will consume fish, frogs and clams, however, when typical plant food sources are scarce (Environment Canada 2000). The diet of the marsh rice rat is typically made up of equal parts animal and vegetable matter (NSRL 2002). Foods include green vegetation, fungus, and the seeds of sedges, marsh grasses, rice, insects, fiddler crabs, snails, fishes, and the carcasses of small rodents and birds.

For this assessment, the hypothetical omnivorous mammal receptor will be assumed to have a diet that is half animal and half vegetable matter, with the animal portion equally divided between fish, terrestrial invertebrates, and aquatic invertebrates (16.7% each). These values are point estimates with no distribution.

Gross Energy of Prey (GE)

Gross energies of fish and invertebrates, which are dietary food items consumed by omnivorous mammals, were available from the literature. The gross energies of these organisms were reported as follows: bony fish = 1200 kcal/kg (SD = 240; Thayer *et al*. 1973); bivalves = 800 Kcal/kg (estimated SD = 160; Cummins and Wuycheck 1971), crabs = 1000 Kcal/kg (SD = 210; Thayer *et al*. 1973), and shrimp (1100

Kcal/kg (SD = 240; Cummins and Wuycheck 1971). For aquatic invertebrates consumed by omnivorous mammals, the mean gross energy was set to 967 Kcal/kg (estimated standard deviation = 193) in the Monte Carlo analysis. The distribution for this variable was assumed to be lognormal. Incertitude was considered low for this input variable because: (1) sufficient experimental data were available to confidently estimate the mean and standard deviation, (2) the variable is easily measured and thus measurement error is low, and (3) there appears to be little difference in the gross energies of different prey species. Therefore, probability bounds were not derived for this variable.

Assimilation Efficiency of Prey (AE)

The assimilation efficiency of omnivorous mammals depends upon the animal itself as well as the type of diet (USEPA 1993). Diets that include high percentages of indigestible material, such as ash, chitin or cellulose, tend to have lower assimilation efficiencies. Prey items with higher percent body weight fat will have higher assimilation efficiencies. The estimated assimilation efficiency for fish consumed by mammals is 91% (estimated SD = 9.0; USEPA 1993, estimated from Nagy 1987). The assimilation efficiency for the consumption of aquatic invertebrates (crabs and bivalves) by mammals was not found. However, small mammals consuming bivalves would shell the prey prior to consumption, so we can assume an efficiency similar to that of fish (mean = 91%; SD = 9.0). Beta distributions were assumed for this variable with parameters of: alpha = 60; beta = 7; scale = 1.0. This parameterization results in a distribution that has a mean close to 91%. With this distribution, there is approximately a 95% probability that assimilation efficiency will be between 81 and 96%. Incertitude was considered low for this input variable because: (1) the variable is easily measured and thus measurement error is low, and (2) there appears to be little difference in the gross energies of different prey species. Therefore, probability bounds were not derived for this variable.

Concentrations of COCs in Prey Items

Fish prey consumed by omnivorous mammals are assumed to be of group 1, 2a, and 2b and include gulf killifish, gulf menhaden, sheepshead minnow, spot and sunfish. Total mercury concentrations were used as a surrogate for methylmercury when methylmercury concentrations were not available. In fish tissues, however, Bloom (1992) found that methylmercury accounted for close to 100% of total mercury.

Concentrations of methylmercury were available for invertebrate groups 1a and 1b (*Rangia* and hermit crab). For group 2a (shrimp), total mercury concentrations were used as a surrogate for methylmercury when methylmercury concentrations were not available. An analysis of samples having both measured concentrations of methylmercury and total mercury showed that methylmercury comprises a large part of total mercury for 2a invertebrates. In 87% of these samples, methylmercury concentrations were within 20% of total mercury concentrations. Some of the discrepancy might be due to experimental error.

Concentration of Methylmercury in Fish

Bayou d'Inde AOC

A number of group 1, 2a, and 2b fish species were sampled in the Bayou d'Inde AOC, including gulf killifish, gulf menhaden, and spot. These were analyzed for mercury concentrations. The mean was derived by fitting the data to a lognormal distribution using Crystal Ball 2000 (Decisioneering 2000) which gave a fitted mean of 0.189 mg/kg ww and a standard deviation of 0.193 (n = 103). During long exposures, omnivorous mammals will spatially and temporally average their exposures. To represent this averaging, we used a bootstrapping process to sample from the mercury in fish distribution over a period that includes the reproductive cycle of most omnivorous mammals. Thus, omnivorous mammals were assumed to forage over 160 days, and each day consume fish having a lognormal mercury

distribution with a mean of 0.189 mg/kg ww and standard deviation of 0.193. The resulting grand mean for the 160 days was 0.189 mg/kg ww and the standard deviation on the grand mean was 0.00638. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 1, 2a, and 2b fish species were sampled in the reference areas of the Calcasieu Estuary, including gulf killifish, sheepshead minnow and mullet. These were analyzed for mercury concentrations. The fitted mean was 0.0244 mg/kg ww with a standard deviation of 0.00968 (n = 18). The grand mean for the 160 days calculated using the bootstrapping technique was 0.0244 mg/kg ww with a standard deviation of 0.000306. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Concentration of Methylmercury in Aquatic Invertebrates

Bayou d'Inde AOC

A number of group 1a, 1b, and 2a invertebrate species were sampled in the Bayou d'Inde AOC, including *Rangia*, fiddler crabs, and shrimp. These were analyzed for mercury concentrations. The fitted mean was 0.0373 mg/kg ww with a standard deviation of 0.0123 (n = 22). The grand mean for the 160 days calculated using the bootstrapping technique was 0.0373 mg/kg ww with a standard deviation of 0.000434. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95%

confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 2a invertebrate species were sampled in the reference areas of the Calcasieu Estuary, primarily shrimp. These were analyzed for mercury concentrations. The fitted mean was 0.00751 mg/kg ww with a standard deviation of 0.00174 (n = 10). The grand mean for the 160 days calculated using the bootstrapping technique was 0.00751 mg/kg ww with a standard deviation of 0.0000587. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Concentration of TCDD-TEQs in Fish

Bayou d'Inde AOC

A number of group 1, 2a, and 2b fish species were sampled in the Bayou d'Inde AOC, including gulf menhaden, gulf killifish and spot. These were analyzed for TCDD-TEQs concentrations. The fitted mean was 29.6 ng/kg ww with a standard deviation of 14.4 (n = 24). The grand mean for the 160 days calculated using the bootstrapping technique was 29.6 ng/kg ww with a standard deviation of 0.459. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Middle Calcasieu River AOC

A number of group 1, 2a, and 2b fish species were sampled in the Middle Calcasieu River AOC, including sheepshead minnow, anchovy, and spot. These were analyzed for TCDD-TEQs concentrations. The fitted mean was 16.3 ng/kg ww with a standard deviation of 14.8 (n = 5). The grand mean for the 160 days calculated using the bootstrapping technique was 16.23 ng/kg ww with a standard deviation of 0.434. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic calculation was not used as there were only five samples and the coefficient of variation for the Land statistic was greater than one. A uniform distribution was used as an alternative approach for estimating the upper and lower bounds on the mean.

Reference Areas

A number of group 1, 2a, and 2b fish species were sampled in the reference areas of the Calcasieu Estuary, including gulf killifish and mullet. These were analyzed for TCDD-TEQs concentrations. The fitted mean was 7.64 ng/kg ww with a standard deviation of 2.89 (n = 4). The grand mean for the 160 days calculated using the bootstrapping technique was 7.64 ng/kg ww with a standard deviation of 0.119. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic calculation was not used as there were only four samples and the coefficient of variation for the Land statistic was greater than one. A uniform distribution was used as an alternative approach for estimating the upper and lower bounds on the mean.

Concentration of TCDD-TEQs in Aquatic Invertebrates

Bayou d'Inde AOC

A number of group 1 and 2a invertebrate species were sampled in the Bayou d'Inde AOC, including mussels and shrimp. These were analyzed for TCDD-TEQs

concentrations. The fitted mean was 22.3 ng/kg ww with a standard deviation of 14.4 (n = 3). The grand mean for the 160 days calculated using the bootstrapping technique was 22.2 ng/kg ww with a standard deviation of 0.556. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic calculation was not used as there were only three samples and the coefficient of variation for the Land statistic was greater than one. A uniform distribution was used as an alternative approach for estimating the upper and lower bounds on the mean.

Middle Calcasieu River AOC

A number of group 2a invertebrate species were sampled in the Middle Calcasieu River AOC, primarily shrimp. These were analyzed for TCDD-TEQs concentrations. The fitted mean was 7.29 ng/kg ww with a standard deviation of 0.642 (n = 3). The grand mean for the 160 days calculated using the bootstrapping technique was 7.29 ng/kg ww with a standard deviation of 0.0230. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic calculation was not used as there were only three samples and the coefficient of variation for the Land statistic was greater than one. A uniform distribution was used as an alternative approach for estimating the upper and lower bounds on the mean.

Reference Areas

No aquatic invertebrate samples were available from the reference areas of the Calcasieu Estuary, therefore the TCDD-TEQs tissue concentration in these species in this region were assumed to be zero.

Concentration of Selenium in Fish

Bayou d'Inde AOC

A number of group 1, 2a, and 2b fish species were sampled in the Bayou d'Inde AOC, including gulf menhaden, gulf killifish and sheepshead minnow. These were analyzed for selenium concentrations. The fitted mean was 0.563 mg/kg ww with a standard deviation of 0.198 (n = 103). The grand mean for the 160 days calculated using the bootstrapping technique was 0.562 mg/kg ww with a standard deviation of 0.00620. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Middle Calcasieu River AOC

A number of group 1, 2a, and 2b fish species were sampled in the Middle Calcasieu River AOC, including gulf killifish, sheepshead minnow and spot. These were analyzed for selenium concentrations. The fitted mean was 0.502 mg/kg ww with a standard deviation of 0.482 (n = 30). The grand mean for the 160 days calculated using the bootstrapping technique was 0.499 mg/kg ww with a standard deviation of 0.0133. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Upper Calcasieu River AOC

A number of group 1, 2a, and 2b fish species were sampled in the Upper Calcasieu River AOC, including gulf killifish, gulf menhaden and bay anchovies. These were analyzed for selenium concentrations. The fitted mean was 0.510 mg/kg ww with a standard deviation of 0.198 (n = 51). The grand mean for the 160 days calculated

using the bootstrapping technique was 0.509 mg/kg ww with a standard deviation of 0.00625. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 1, 2a, and 2b fish species were sampled in the reference areas of the Calcasieu Estuary, including gulf killifish, sheepshead minnow and mullet. These were analyzed for selenium concentrations. The fitted mean was 0.305 mg/kg ww with a standard deviation of 0.180 (n = 18). The grand mean for the 160 days calculated using the bootstrapping technique was 0.304 mg/kg ww with a standard deviation of 0.00571. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Concentration of Selenium in Aquatic Invertebrates

Bayou d'Inde AOC

A number of group 1a, 1b and 2a invertebrate species were sampled in the Bayou d'Inde AOC, including *Rangia*, fiddler crab, and shrimp. These were analyzed for selenium concentrations. The fitted mean was 0.460 mg/kg ww with a standard deviation of 0.0434 (n = 22). The grand mean for the 160 days calculated using the bootstrapping technique was 0.460 mg/kg ww with a standard deviation of 0.00140. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Middle Calcasieu River AOC

A number of group 2a invertebrate species were sampled in the Middle Calcasieu River AOC, primarily shrimp. These were analyzed for selenium concentrations. The fitted mean was 0.464 mg/kg ww with a standard deviation of 0.175 (n = 20). The grand mean for the 160 days calculated using the bootstrapping technique was 0.463 mg/kg ww with a standard deviation of 0.00551. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Upper Calcasieu River AOC

A number of group 1a, 1b, and 2a invertebrate species were sampled in the Upper Calcasieu River AOC, including *Rangia*, fiddler crab, and shrimp. These were analyzed for selenium concentrations. The fitted mean was 0.575 mg/kg ww with a standard deviation of 0.155 (n = 33). The grand mean for the 160 days calculated using the bootstrapping technique was 0.575 mg/kg ww with a standard deviation of 0.00505. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 2a invertebrate species were sampled in the reference areas of the Calcasieu Estuary, primarily shrimp. These were analyzed for selenium concentrations. The fitted mean was 0.424 mg/kg ww with a standard deviation of 0.186 (n = 10). The grand mean for the 160 days calculated using the bootstrapping technique was 0.423 mg/kg ww with a standard deviation of 0.00594. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the

Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Concentration of Total PCBs in Fish

Bayou d'Inde AOC

A number of group 1, 2a, and 2b fish species were sampled in the Bayou d'Inde AOC, including gulf killifish, gulf menhaden, and spot. These were analyzed for total PCB concentrations. The fitted mean was 0.158 mg/kg ww with a standard deviation of 476 (n = 102). The resulting grand mean for the 160 days was 0.162 mg/kg ww and the standard deviation on the grand mean was 0.0148. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 1, 2a, and 2b fish species were sampled in the reference areas of the Calcasieu Estuary, including gulf killifish, sheepshead minnow and mullet. These were analyzed for total PCB concentrations. The fitted mean was 0.0224 mg/kg ww with a standard deviation of 0.0904 (n = 17). The grand mean for the 160 days calculated using the bootstrapping technique was 0.0230 mg/kg ww with a standard deviation of 0.00258. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Concentration of Total PCBs in Aquatic Invertebrates

Bayou d'Inde AOC

A number of group 1a, 1b, and 2a invertebrate species were sampled in the Bayou d'Inde AOC, including mussels, shrimp, and fiddler crab. These were analyzed for total PCB concentrations. The fitted mean was 0.0329 mg/kg ww with a standard deviation of 0.0264 (n = 37). The grand mean for the 160 days calculated using the bootstrapping technique was 0.0328 mg/kg ww with a standard deviation of 0.000732. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

Reference Areas

A number of group 1a and 2a invertebrate species were sampled in the Bayou d'Inde AOC, primarily mussels and shrimp. These were analyzed for total PCB concentrations. The fitted mean was 0.0101 mg/kg ww with a standard deviation of 0.0100 (n = 13). The grand mean for the 160 days calculated using the bootstrapping technique was 0.0100 mg/kg ww with a standard deviation of 0.000268. This variable was assumed to be lognormally distributed. For the probability bounds analysis, the Land statistic was used to determine the lower and upper 95% confidence limits on the mean. The resulting values were used to parameterize a lognormal distribution.

2.2.1.3 Monte Carlo Analysis

The Monte Carlo analyses for exposure combined the input distributions as per Equation 1, described in Section 2.3.1. The input distributions are summarized in

Table I1-2. Each analysis included 10,000 trials and Latin Hypercube Sampling to ensure adequate sampling from all portions of the input distributions. The analyses were done in Crystal Ball 2000 (Decisioneering 2000). Considering all possible pairwise combinations of input variables, no dependencies were expected. Therefore, no correlations were included in the Monte Carlo analyses. The Monte Carlo analyses made no distinction in the way incertitude and variability were propagated; they were simply combined. We addressed incertitude and variability separately in the probability bounds analyses described below.

2.2.1.4 Probability Bounds Analysis

The probability bounds analyses were run using RiskCalc, version 4.0 (Ferson 2002). For the probability bounds analyses, we used a similar table to that of the input variables used for the Monte Carlo analyses. With the exception of tissue concentrations, the input variables are similar to those used in the Monte Carlo analyses. Mean tissue concentrations used in the probability bounds analyses were calculated using the Land statistic. The input distributions are summarized in Table I1-3.

2.2.2 Effects Characterization

The purpose of this section is to: (1) briefly review the literature on the effects of dietary methylmercury, selenium, total PCBs, and TCDD-TEQs to omnivorous mammals, and (2) select the appropriate effects metric for each COC to be used with the results of the exposure assessment to estimate risk. We will focus on ecologically relevant effects endpoints such as survival, growth, and reproduction. Examples of omnivorous mammal species considered in this section include raccoons, muskrats,

opossums and marsh rice rats. Because the available toxicological information for these species is limited, data from other mammal studies will be discussed where appropriate. Also, because the category of omnivorous mammals spans several taxonomic groups, the animals considered as surrogates are fairly disparate. Other information on the toxicity of methylmercury, selenium, total PCBs, and TCDD-TEQs to wildlife can be found in Appendices 5 and 10, respectively, of the problem formulation document (MacDonald *et al*. 2001).

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to dose-response curves for the receptor group of interest (i.e., omnivorous mammals). In this assessment, effects characterization will preferentially rely on dose-response curves, but may default to benchmarks or other estimates of effect [e.g., no observed adverse effect level (NOAEL), lowest observed adverse effect level (LOAEL)] when insufficient data are available to derive dose-response curves. Effects associated with survival, growth, and reproduction are generally the preferred measures of effect. The objective of this section is to provide an overview of the available options for characterizing effects information and to describe the decision criteria for choosing among them for omnivorous mammals exposed to COCs.

The following is the hierarchy of decision criteria used to characterize effects for each receptor group-COC combination:

1. Had bioassays with five or more treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, we estimated the dose-response relationship using the Generalized Linear Model (GLiM) framework described in Kerr and Meador (1996) and Bailer and Oris (1997). The GLiM framework involves conducting linear regression analysis on dose-response data that have

been transformed to linearize the relationship (e.g., probit transformation for survival data). If not, we proceeded to 2.

2. Were multiple bioassays available that, when combined, had five or more treatments on the receptor group of interest or a reasonable surrogate? Such bioassays would be expected to have had similar protocols, exposure scenarios and effects metrics. If yes, we estimated the dose-response relationship as in 1. If not, we proceeded to 3.

3. Had bioassays with less than five treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, we conducted hypothesis testing to determine the NOAEL and LOAEL or reported these metrics when available from the original study. If not, we proceeded to 4.

4. Were sufficient data available from field studies and monitoring programs to estimate concentrations or doses of COCs consistently associated with no adverse effects and with adverse effects to omnivorous mammals? If yes, we developed field-based no effects and effects measures. This approach is analogous to the approach used to develop sediment-quality guidelines for the protection of aquatic life (see Long *et al*. 1995; MacDonald *et al*. 1996; MacDonald *et al*. 2000b). If not, we proceeded to 5.

5. A range within which the threshold for the receptor group of interest was expected to occur was derived. Because information on the sensitivity of the receptor of interest was lacking, it was difficult to derive a threshold that was neither biased high or low. If bioassay data were available for several other species, however, a threshold range could be calculated that spanned sensitive and tolerant species. This range was assumed to include the threshold for the receptor group of interest.

2.2.2.1 Mercury

Mercury has no known physiological use to mammals, but has teratogenic, mutagenic, and carcinogenic affects (Eisler 2000c). Mercury most commonly exists as methylmercury (MeHg) in higher trophic level species (Wolfe *et al*. 1998). MeHg attacks the central nervous system, affecting coordination, site, hearing, and sensory functions (Eisler 2000c). Acute effects of MeHg include muscular uncoordination, falling, slowness, calmness, and hyperactivity (USEPA 1997b). Chronic exposure may lead to liver or kidney damage, neurobehavioral effects, reduced food consumption, weight loss, impaired growth, effects to reproduction, and death (USEPA 1997b).

Survival

Hoskins and Hupp (1978) investigated the effects of methylmercury to rat, hamster, and squirrel monkey administered via single intraperitoneal injection. Using probit analysis, the 24 hour and 30-day LD_{50}s were determined for each animal. The rat was the most sensitive and the 24 hour LD_{50} was 11.9 mg/kg bw and a 30 day LD_{50} of 10.1 mg/kg bw. The hamster was the next most sensitive with a 24 hour LD_{50} of 19.0 mg/kg bw and a 30 day LD_{50} of 13.3 mg/kg bw. Squirrel monkeys treated with injections of 3.6 - 17.0 mg/kg bw did not die within 24 hours, therefore the 24 hour LD_{50} can only be stated as being greater than 17.0 mg/kg bw. The 30 day LD_{50} for monkeys can be estimated at between 5.6 and 6.4 mg/kg bw, as the 6.4 mg/kg bw dose reduced the animals to such a state of reduced capacity that sacrifice was necessary, and monkeys dosed at 4.8 and 5.6 mg/kg bw survived for the 30 days.

A two week toxicity range-finding test (Verschuuren *et al*. 1976a) exposed weanling Wistar rats to methylmercury chloride at dietary concentrations of 0, 0.1, 0.5, 2.5, 12.5 and 250 mg/kg. Rats receiving the lower concentrations in feed (0-12.5 mg/kg)

showed no signs of toxicity, but animals receiving the highest concentration of MeHgCl in feed (250 mg/kg) showed signs of toxicity after 7 days. These rats had bent backs, piloerection, an unsteady gait, and they died a few days later. Accounting for food intake, this dietary concentration represented a total oral dose of 91 - 103 mg/kg bw and a daily dose of 9.1 - 10.3 mg/kg bw/d. The study was extended over 12 weeks with the lower feed concentrations (and adding a 25 mg/kg level). Food intake was significantly reduced at five weeks in the 25 mg/kg treatment and weight gain was adversely affected in both males and females by week twelve. Verschuuren *et al*. (1976b) then investigated the chronic effects of methylmercury chloride to rats in oral treatments spanning 2 years. Treatment levels ranged from 0 to 2.5 mg/kg in feed, but even at the highest dose, no significant reduction in growth rate was observed in male or female rats.

Male and female Wistar rats in groups of 50 were given diets containing methylmercury such that the daily doses to the animals were 0, 0.002, 0.01, 0.05 or 0.25 mg/kg bw/d for up to 26 months (Munro *et al*. 1980). Rats were in the weanling stage, and weighed 60-70 g at the beginning of the investigation. Body weights and food consumption were monitored over the course of the study. At the highest treatment level, male rats suffered increased mortality, decreased body weights, and pathological changes in the kidneys, peripheral nerves and spinal cord. Kidney damage included enlargement, dilated tubules in the cortex and a granular coating on the outer surface. Neurological toxicity consisted of loss of balance, hind leg crossing and paralysis after 6 months. Body weights of the animals in this test group were generally lower than that of the controls and 16 months into the test, all of the rats had either succumbed or were sacrificed because of their severe condition. At the twelve month interval of the study, 90% of controls were alive, 80% of the 0.002 and 0.05 mg/kg bw/d treatment groups were alive and 60% of the 0.25 mg/kg bw/d treatment group were alive.

Harris *et al*. (1972) injected golden hamsters daily with methylmercuric chloride doses of 1.0, 2.0 or 4.0 mg/kg bw/d on gestation days 1-14. The 2.0 mg/kg bw/d dose caused 20% mortality in dams and the 4.0 mg/kg bw/d dose caused 100% mortality.

Mink fed a daily dose of 0.58 mg/kg MeHg exhibited no obvious signs of mercury poisoning after 25, 50, 75, or 100 d of exposure (Jernelöv *et al*. 1976). Similarly, in a two generation study on mink by Dansereau *et al*. (1999), diets of 0.1 and 0.5 mg/kg total (T) Hg had no effect on survival. However, a dose of 1.0 mg/kg T Hg killed 30 of 50 first generation mink. Neurological and clinical signs of poisoning were observed between 75 and 100 d of exposure. In the second generation, 6 of 7 mink in the 1.0 mg/kg T Hg died after 330 d of exposure. Death usually occurred 7 d after the first clinical signs. Effects included weight loss, lethargy, uncoordination, and splaying of hind legs (Dansereau *et al*. 1999). Wren *et al*. (1987a) also found these effects in male and female mink fed 1.0 mg/kg MeHg for 180 d. Eight of 12 females and 1 of 4 males died during the experiment. Both clinical and pathological signs of poisoning were found. The mean T Hg concentration in tissues of females that died were: liver 44.1, kidney 28.4, and brain 15.3 mg/kg (Wren *et al*. 1987a). The average daily dose was estimated to be 0.18 mg/kg bw/d for females and 0.10 mg/kg bw/d for males (Wren *et al*. 1987a).

The onset of adverse effects occurs more quickly as the dietary dose increases. All mink exposed to dietary levels of 1.1, 1.8, 4.8, 8.3 or 15 mg/kg MeHgCl showed histopathological effects after 93 d of exposure (Wobeser *et al*. 1976). The only clinical sign from mink fed 1.1 mg/kg was a tendency to move more slowly during the last three days of the study. None of the mink from this group died during the experiment. Mink in the 1.8-15 mg/kg dose groups developed clinical signs of Hg poisoning within the experimental period. Clinical signs included anorexia, weight loss, head tremors, ataxia, and convulsions; deaths occurred in all of these groups.

Behavioral effects, such as listlessness and indifference to investigators, were also observed. Mean Hg concentrations in tissues were: brain 11.9, muscle 16.0, and kidney 23.1 mg/kg and Wobeser *et al*. (1976) concluded that brain Hg concentrations ≥5 mg/kg can lead to Hg poisoning.

Aulerich *et al*. (1974) fed mink 5 mg/kg MeHg. Effects to mink were similar to those in the 4.8 mg/kg MeHgCl dose group of Wobeser *et al*. (1976). Clinical signs including anorexia, uncoordination, and convulsions were apparent after a latency period of 24 d and death usually occurred after 33 d of exposure (Aulerich *et al.* 1974). Mercury concentrations in dead mink were similar between dose groups although exposure times were different.

River otters display effects due to mercury similar to those reported in mink. O'Connor and Nielsen (1980) fed river otters either 2, 4 or 8 mg/kg MeHg in their diet. The average survival times for each of the treatment groups was 184, 117, and 54 d, respectively. The onset of intoxication correlated with the dietary concentration and all dosed river otters followed the same toxicological pattern. The central nervous system deteriorated progressively over a 10-14 d period. Initial signs of intoxication were anorexia and lethargy. This was followed by posterior ataxia and splaying of hind legs. As the poisoning progressed, sight became impaired and animals became recumbent. In the latter stages, symptoms included aggressiveness, convulsions and terminal recumbency (O'Connor and Nielsen 1980). Mean T Hg tissue concentrations were: kidney 39, liver 33, muscle 20, and brain 18 mg/kg. As a result of their findings, O'Connor and Nielsen (1980) surmised that adverse subclinical effects to behavior and reproduction could result from prolonged exposure to less than 2 mg/kg MeHg in the diet.

Mattsson *et al*. (1981) found evidence of preclinical effects in 12-14 month old beagle dogs (*Canis familians*) given a daily dose of 0.5 mg/kg MeHg. Effects to the visually evoked response (VER) were observed. Damage to VER can affect hunting ability, reproduction, and the survival of young (Mattsson *et al*. 1981).

Three studies have documented adverse effects in cats given daily oral doses of 0.25 mg MeHg/kg bw/d (Charbonneau *et al*. 1974; Eaton *et al*. 1980; Khera *et al*. 1974 in Khera 1979). Charbonneau *et al*. (1974) orally dosed 8 cats with gelatin capsules containing methylmercuric chloride dissolved in corn oil. A second group of 4 cats was fed a mercury contaminated fish diet so that the animals received the 0.25 mg MeHg/kg bw/d dose. No significant differences were found in the onset of MeHg intoxication between the groups. Both groups showed clinical signs of intoxication between 55 and 96 d. Symptoms included ataxia, tremors, impaired righting reflex, and convulsions. Pathological examinations revealed lesions in the cerebellar vermis and cortex (Charbonneau *et al*. 1974). In comparison, Eaton *et al*. (1980) first saw neurological abnormalities on day 68 of a 90 d study. All cats in the study displayed signs of intoxication by the end of the exposure period. The mean survival time was 78 d and symptoms of intoxication were similar to those reported in Charbonneau *et al*. (1974). Khera *et al*. (1974) dosed newborn kittens with 0.25-1.0 mg/kg MeHgCl for up to 184 d. Kittens developed toxicoses followed by degenerative changes in granule cells, Purkinje cells, and cerebral neurons. Interestingly, kittens required a higher daily dose than adults to elicit symptoms of toxicity (Khera *et al*. 1974).

No laboratory studies were found for raccoons or other omnivorous mammals found in the Calcasieu Estuary. The studies discussed for mink, river otters, and other species reported that effects occurred to these species at dietary levels of 1-2 mg/kg (Dansereau *et al*. 1999; Wobeser *et al*. 1976). Central nervous system function was the most common target. Animals usually become anorexic and lethargic. This was

followed by muscular ataxia and uncoordination, recumbency, convulsions and death. Methylmercury is lethal to river otters at a dietary concentration of 2.0 mg/kg (O'Connor and Nielsen 1980). Cats appear to be more sensitive to the adverse effects of MeHg than mink or river otters.

Reproduction

Methylmercury can have adverse effects to young mammals at levels considered harmless to adults (Eisler 2000c). All forms of Hg can cross the placenta, but MeHg specifically concentrates in the fetal brain. Reproductive effects resulting from exposure to MeHg include developmental alterations leading to behavioral impairments after birth, as well as decreased fertility and increased occurrence of fetal death (Eisler 2000c).

Verschuuren *et al*. (1976c) conducted a three-generation study of the reproductive effects of methylmercury chloride to Wistar rats. Twenty female and ten male weanling rats were maintained on diets of 0, 0.1, 0.5, and 2.5 mg/kg methylmercuric chloride for 2 years. At six weeks, each male was permitted to mate with 2 females to produce the F1a litter and 6 weeks later, mating was repeated to produce the F1b generation. These litters were then mated to produce the F2 generation. No significant histological changes were observed at any treatment level, nor were any effects observed on the fertility index (pregnancy/matings), lactation index (pups alive at day 21/pups alive at day 5), or 21 day body weight gain of pups. The viability index (pups alive at day 5/pups born), however, was significantly reduced in the F1 and F2 generations in the 2.5 mg/kg treatment level. This represents an oral dose of 0.16 mg/kg bw/d (Sample *et al*. 1996).

Pregnant Fischer 344 rats orally dosed with 10, 20, or 30 mg/kg bw methylmercuric chloride on day 7 of gestation were observed for toxic effects, body burdens, and

body weights of dams and fetuses. Fetal survival rates were 19.2, 41.4, and 91.1% lower than controls, respectively. Preimplantation loss was 5.9, 13.7 and 22.5%, respectively, while post implantation loss was 16.7, 34.1, and 88.9%, respectively for the three treatment groups. The LD_{50} for fetal loss in Fischer 344 rats was calculated to be 16.5 mg/kg bw for single oral doses of methylmercuric chloride (Lee and Han 1995). Female Swiss-Webster mice were given daily doses of 0.001, 0.01, 0.1, 1.0, 5.0 or 10.0 mg/kg bw/d by gavage from day 6 to 17 of gestation (Khera and Tabacova 1973). Mice given the 5.0 mg/kg bw/d treatment produced no litters and all dams on the 10.0 mg/kg bw/d treatment died during pregnancy. Gates *et al*. (1986) orally dosed mice with methylmercuric chloride on days 9.5, 12.5 and 18.5 of gestation. Treatment levels were 3.6, 5.3, 8.0, 12.0, 18.0, and 27.0 mg/kg bw and were administered by gavage. Rats treated at levels of 12.0 mg/kg bw or higher suffered significant reductions in the proportion of dams with viable pups, the number of viable pups per litter, and the percent viable pups per litter.

Khera (1973) investigated the reproductive effects of methylmercuric chloride in male Sprague-Dawley rats. Animals were treated by gavage with 1.0, 2.5, or 5.0 mg/kg bw/d for 5-7 days, then mated with untreated females. A dose-related reduction in mean litter size was observed following the treatments. Friedmann *et al*. (1998) dosed adult male brown Norway rats with 0.0008, 0.008, and 0.08 mg/kg bw/d over a period of 19 weeks and a dose related accumulation of mercury in the testes was observed. Effects of mercury exposure included decreased testosterone levels, decreased cauda epididymal sperm storage, and decreased male fertility. The decrease in male fertility was measured in terms of the number of pregnant females per successful mating. This value was 5/14 for the control group, but fell to 0/13 in the highest treatment group.

Khera and Tabacova (1973) investigated the reproductive effects of methylmercuric chloride in female Wistar rats. Rats were placed on diets containing methylmercuric chloride such that the doses received equaled 0.002, 0.01, 0.05, or 0.25 mg/kg bw/d for 122 days. These treatment levels had no observable adverse effects to female rats in terms of weight gain, behavior, or pregnancy. Fuyuta *et al*. (1978) observed methylmercuric chloride to significantly reduce growth of Wistar rat offspring when dams were treated at levels of 4.0 mg/kg bw/d on days 7 through 14 of gestation. A dose of 6.0 mg/kg bw/d also reduced pup growth rate, although not significantly at $p = 0.01$. Incidences of malformation were also observed in this study to be significantly more numerous in the higher treatment levels than in the 2.0 and 0 mg/kg bw/d doses. Suter and Schon (1986) treated HAN-Wistar rats with methylmercuric chloride in drinking water at levels equivalent to 0.21, 0.75, and 1.6 mg/kg bw/d for a period spanning 13 days before mating to 21 days after giving birth. At the lowest dose, a number of adverse developmental effects were observed, including delayed vaginal opening, impaired midair righting reflex, and impaired swimming ability. At the 1.6 mg/kg bw/d treatment level, a high incidence of pup mortality was observed, as well as some signs of toxicity in the dams.

Sprague-Dawley albino rats were dosed with 0, 0.2, 1.0, 2.0, and 4.0 mg/kg bw/d methylmercuric chloride during gestation days 6 through 15 in a study conducted by Geyer *et al*. (1985). No effects were observed in the pups of dams dosed at 1.0 mg/kg bw/d or less, while pups of the 2.0 mg/kg bw/d treatment group had significantly reduced body weights, developmental abnormalities, and reduced surface righting ability. Rats in the 4.0 mg/kg bw/d treatment group produced no live offspring. Vorhees (1985) dosed pregnant Sprague-Dawley rats at 1.6 and 4.8 mg/kg bw/d methylmercuric chloride on days 6 to 9 of gestation. The lower treatment level produced no adverse reproductive effects on the pups while the 4.8 mg/kg bw/d dose

lengthened the gestation period, increased pre-weaning mortality of the pups, and reduced offspring weight at 60 days.

Wren *et al*. (1987b) observed no effect on male mink fertility, percentage of females whelped, or number of kits born from adults fed a diet containing 1.0 mg/kg MeHg. Dansereau *et al*. (1999) similarly found that although the proportion of females giving birth was significantly different between dose groups (0.1 - 93.7%, 0.5 - 52.0%, 1.0 mg MeHg/kg - 33.0%). Other endpoints such as gestation period and litter size were not significantly different.

Placental transfer of Hg was demonstrated by Wren *et al*. (1987a). Mink kits born from adults fed 1.0 mg/kg MeHg contained high levels of Hg on the day of whelping. At 5 weeks of age, these levels had decreased, suggesting that Hg transfer through mothers milk is not a significant route of transport.

Adult dogs and cats dosed with 0.1-0.25 mg/kg of MeHg chloride during pregnancy demonstrated a variety of reproductive effects, such as increases in abortion, stillbirths, and irregular fetuses (Earl *et al*. 1973; Khera 1973). High incidence of stillbirths was also recorded in sows fed up to 0.5 mg/kg MeHg during pregnancy (Earl *et al*. 1973; Khera 1973).

Field Surveys

Field studies indicate a correlation between Hg concentration in prey and predator species. They also illustrate local and regional variation of Hg exposure. This is especially true for the transfer and biomagnification of Hg from aquatic systems to piscivorous wildlife (Wren *et al*. 1986; Sheffy and St. Amant 1982). The field studies discussed below focus on clinical symptoms of poisoning, pathological and/or histological lesions found, and the tissue concentrations from collected animals.

Wobeser and Swift (1976) examined a wild mink captured near the South Saskatchewan River that had been found acting strangely and which died shortly after capture. The mink appeared in good condition, however histological lesions were found similar to experimentally dosed mink. Hg tissue concentrations were: liver 58.3, kidney 31.9, muscle 15.2, and brain 13.4 mg/kg Hg.

Sheffy and St. Amant (1982) analysed a variety of small mammals trapped in Wisconsin between 1972-75. Their study found that otters had the highest Hg burdens followed by mink > raccoon > fox > muskrat > beaver. Mean Hg tissue concentrations for mink were: kidney 2.33, liver 2.08, and brain 0.46 mg/kg. The maximum Hg tissue concentrations in 39 mink collected were: fur 41.2, kidney 12.5, and liver 17.4 mg/kg. Sheffy and St. Amant (1982) found Hg tissue concentrations were higher from industrialized sections of the Wisconsin River than non-industrial areas. Osowski *et al*. (1995) found a similar pattern in tissue concentrations. Declining mink populations from coastal areas of Georgia and North Carolina had mean Hg concentrations in their kidneys of 2.24 mg/kg. In contrast, mink from the Piedmont regions had mean Hg concentrations of 0.53 mg/kg.

O'Connor and Nielsen (1980) examined mink harvested by trappers in the northeastern US. Histological and pathological examinations found lesions in some of the mink. Forty-four percent of the mink examined also had lesions in their central nervous system. Mean liver Hg concentration was higher in males (1.20 mg/kg) than females (0.73 mg/kg; O'Connor and Nielsen 1980).

Foley *et al*. (1988) reported tissue concentrations for mink from across New York State. Mercury concentrations in liver ranged from 0.25 to 7.66 mg/kg. Wren *et al*. (1986) discovered changes in concentration relative to the proximity of a contaminated site. Mean liver Hg concentrations in a contaminated area were 3.75

mg/kg versus 1.13 mg/kg in samples ≥2 km away. Total Hg concentrations did not differ significantly between males and females. A relationship was found between Hg concentrations in mink and their prey (Wren *et al*. 1986) where tissue burdens in different study areas ranked similarly for mink, crayfish, and fish.

Field studies of river otters also found concentration changes with proximity to contaminated sites (Foley *et al*. 1988; Wren *et al*. 1986) and a correlation between levels in prey species and predators (Wren *et al*. 1986). Wren *et al*. (1986) sampled river otters from five study areas in Ontario. Mean liver Hg concentrations from a contaminated and uncontaminated area were 4.57 mg/kg and 1.3 mg/kg, respectively. The highest liver Hg concentrations were 14.3 and 17.4 mg/kg. The river otter with the former concentration also had a brain Hg concentration of 7.1 mg/kg. Wren *et al*. (1986) also found concentration changes in prey from contaminated to uncontaminated sites. They did not find differences in Hg levels between males and females.

Sheffy and St. Amant's (1982) study of furbearers in Wisconsin recorded the following mean Hg concentrations for river otters: kidney 8.47, liver 3.34, and brain 0.74 mg/kg. Maximum Hg concentrations from 41 river otters were: fur 63.2, kidney 20.9, and liver 23.6 mg/kg. O'Connor and Nielsen (1980) reported a lower mean liver Hg concentration in river otters from the northeastern US. They also found males had a higher mean liver Hg concentration (males 2.14, females 1.12 mg/kg). Approximately half of the river otters examined had lesions in either the lungs, intestines, or bladder (O'Connor and Nielsen 1980).

Raccoons harvested by trappers over four seasons from the Wisconsin River watershed had mean tissue concentrations of: liver 2.01, kidney 1.36, muscle 0.08, and brain <0.02 (Sheffy and St Amant 1982). Raccoons and other piscivorous

animals, such as mink and river otters, were found to have higher Hg levels than herbivorous animals. Roelke *et al*. (1991) reported muscle concentrations from 0.2 to 2.4 mg/kg (cited in Duvall and Barron 2000). Raccoons sampled from near Parry Sound, Ontario had mean Hg concentrations of: liver 4.5, kidney 1.1, and muscle 0.28 mg/kg (Wren *et al*. 1980).

In summary, field studies confirmed that Hg concentrations vary with location and proximity to contaminated sites. Bioaccumulation and biomagnification processes are also evident through the correlation of prey and predator Hg levels. O'Connor and Nielsen (1980) found that males had higher concentrations than females while Wren *et al*. (1986) found no discrepancy. Although animals in the wild may appear healthy and show no clinical signs of Hg toxicity, lesions to tissues and the central nervous system may exist.

Effects Metrics

Toxic responses to methylmercury exposure generally increase with the duration of the exposure and reproductive and developmental effects generally occur at lower doses than mortality. No studies, however, were found that had five or more treatment levels and that investigated a suitably sensitive endpoint such as reproductive impairment to omnivorous mammals or a reasonable surrogate.

The next preferred option is to combine multiple bioassays that, when taken together, provide five or more treatments to the receptor of interest or a surrogate. Two studies (Fuyuta *et al*. 1978; Khera and Tabacova 1973) treated mice with oral doses of methylmercuric chloride given by gavage during gestation. The two studies varied slightly in exposure duration (8 days and 12 days). For this effects metrics section, the adverse reproductive effects of the treatments will be expressed in terms of total methylmercuric chloride averaged over 12 days of gestation. Generally, there were

no significant impacts on the number of live fetuses per female at doses under 2.0 mg/kg bw/d, but effects rose sharply to 100% mortality as the dose exceeded 5.0 mg/kg bw/d.

We fitted the dose-response data from the above two studies to a generalized linear model (GLiM) using SAS® (SAS Institute, Cary, NC). Fecundity data were subjected to a log-link function and a Poisson error distribution was assumed (Bailer and Oris 1997). The model parameters were $\beta_0 = 2.31$, $\beta_1 = -0.292$, se$\beta_0 = 0.134$, se $\beta_1 = 0.0760$, and corr$\beta_0\beta_1 = -0.509$. The resulting dose-response curve for the reproductive effects of methyl mercuric chloride to mice produced an adequate model to fit the data ($F = 33.09$; $p < 0.0001$) and is shown in Figure I1-4.

2.2.2.2 TCDD-TEQs

Coplanar PCDDs, PCDFs, and PCBs act by the same mode of toxic action, initiated by binding to the aryl hydrocarbon receptor protein (Bosveld and van den Berg 1994). The response of organisms can range from mortality (Safe 1994; Eisler and Belisle 1996; Tillitt *et al*. 1996) to enzyme induction (Aulerich *et al*. 1985). The most toxic PCDD and PCDF congeners tend to be those chlorinated in the 2,3,7, and 8 positions, such as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), as these best fit the receptor site. The toxic response to this group of chemicals is therefore related to the three-dimensional structure of the substance, including the degree of chlorination and positions of the chlorine on the aromatic frame. Substances that are more structurally similar to TCDD will elicit a toxic response closer to that of TCDD. The toxicity of PCBs, PCDDs, and PCDFs may therefore be expressed in terms of 2,3,7,8-TCDD Toxic Equivalents (TEQs), as described by van den Berg *et al*. (1998), where the toxicity of the members of this chemical class are all expressed relative to TCDD for

fish, birds, and mammals. This approach is described in further detail in Appendix G. We will use this approach to convert the toxicity test results for PCDD, PCDF, and PCB congeners described below.

Survival

Pohjanvirta *et al*. (1993) investigated the acute oral toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (PeCDD) and 1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin (HxCDD) to Long-Evans (LE) rats and Han/Wistar (H/W) rats given single doses by gavage. TCDD was the most toxic of the chemicals, producing LD_{50}s of 9,800 and 17,700 ng/kg bw for female and male LE rats, respectively. The LD_{50}s for the H/W rats, conversely, were in excess of 7,200,000 ng/kg bw. PeCDD treatments revealed similar strain differences, with female LE rats having LD_{50}s of 20,000-60,000 ng TEQ/kg bw and female H/W rats having LD_{50}s over 1,620,000 ng TEQ/kg bw. HxCDD showed less of a strain-related difference in toxicity with a LD_{50} for H/W female rats of 187,100 ng TEQ/kg bw compared to between 12,000-36,000 ng TEQ/kg bw for LE rats.

Sprague Dawley rats were investigated by Stahl *et al*. (1992) with a similar complement of chemicals: TCDD, PeCDD, HxCDD and 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin (HpCDD). The reported single dose LD_{50}s for these substances were 43,000, 206,000, 88,700 and 63,250 ng TEQ/kg bw, respectively. Van Miller *et al*. (1977) maintained Sprague-Dawley rats on diets containing 1 to 1,000,000 ng/kg TCDD (estimated doses of 0.0428 to 71,400 ng/kg bw/d) for 78 weeks. Dosage levels above 3,400 ng/kg bw/d produced 100% mortality in the test animals within 3 weeks and 57.1 ng/kg bw/d produced 100% mortality within 31 weeks. Kociba *et al*. (1978) treated male and female Sprague Dawley rats with oral doses of TCDD of 0, 1, 10 and 100 ng/kg bw/d for two years. No significant effects were noted below 10 ng TEQ/kg bw/d, but at 100 ng/kg bw/d, a cumulative increase

in mortality ($p<0.05$) in the latter half of the study period was observed, as well as a decrease in mean body weight ($p<0.05$) from 6-24 months compared to controls.

Hochstein *et al*. (1988) randomly separated 16 mink into four groups of four animals each and administered single oral doses of 2,3,7,8-TCDD at levels of 0, 2,500, 5,000, and 7,500 ng/kg bw for 28 days. The mink exhibited a dose-related decrease in feed consumption and also experienced a dose-related decrease in body weight. Other effects from the higher doses included enlarged organs (relative to body weight), such as heart, brain and thyroid as well as discoloration and blotching of the liver and kidneys. Control animals and those dosed with 2,500 ng/kg bw all survived to the conclusion of the 28-day study, but those treated with 5,000 and 7,500 ng/kg bw survived, on average, to 12.3 and 9.5 days, respectively. The LD_{50} for the 28 day single oral dose of TCDD was calculated to be 4,200 ng/kg bw. Mature female mink maintained on a diet of 1,400 ng/kg TCDD (Hochstein *et al*. 2001) for 131 days showed some mortality, experienced significant weight loss, appeared lethargic, and passed bloody stools.

Neonatal mink given daily intraperitoneal injections of TCDD for the first 12 days after birth (Aulerich *et al*. 1988) were treated with doses of 100 and 1,000 ng/kg bw/d. Animals in the higher treatment level did not survive past two weeks while mink in the lower dose group began to lose weight at the second week and experienced 62% mortality by the nineteenth week.

Aulerich *et al*. (1987) conducted mink dietary exposure studies to PCBs and found that dietary PCB 169 concentrations of 50,000 ng/kg (5,000 ng TEQ/kg TEQ diet) were sufficient to cause significant weight loss and mortality in adult female mink exposed over a period of 135 days. A previous study (Aulerich *et al*. 1985) showed 100% mink mortality within 60 days when fed a diet containing 500,000 ng/kg PCB

169 (50,000 ng TEQ/kg diet) and 50% mortality in a span of 3 months when fed a diet containing 100,000 ng/kg (10,000 ng TEQ/kg diet TEQ).

Reproduction

Holtzman rats exposed to TCDD at a dose of 1,000 ng/kg bw/day on day 15 of gestation experienced a 19% decrease in fetal survival ($p<0.05$) to day 21 of gestation (Mably *et al*. 1992). Similarly, Giavini *et al*. (1983) treated rats to doses of 2,000 ng/kg bw/day and observed that fetal survival to 21 days of gestation decreased by 45% ($p<0.01$) compared to controls. Khera and Ruddick (1973) dosed pregnant Wistar rats at TCDD levels of 0, 125, 250, 500, 1,000, 2,000, 4,000, 8,000, 16,000 ng/kg bw/day on gestation days 6 to 15. At day 22, the animals were sacrificed and it was found that doses above 4,000 ng/kg bw/day produced 100% embryonic lethality. Huuskonen *et al*. (1994) treated Long-Evans rats to an oral dose of TCDD of 5,000 ng/kg bw/day on day 8 of gestation and observed a significant ($p<0.05$) decrease in the number of living fetuses per litter. Over 70% of implantations were resorbed while 5% died at a later fetal stage. Similar observations were made by Sparschu *et al*. (1971) in Sprague Dawley rats fed doses of TCDD of 0, 30, 125, 500, 2,000 and 8,000 ng/kg bw/day on days 6-15 of gestation. Pregnancy was terminated on day 20 of gestation by decapitation. The number of viable fetuses decreased and the total number of resorptions increased dose dependently starting at 500 ng/kg bw/day. Maternal body weight gains decreased ($p< 0.01$) at 500 ng/kg bw/day while fetal body weight decreased ($p<0.01$) at 125 ng/kg bw/day. Pre- and post-implantation loss due to TCDD treatment were observed by Giavini *et al*. (1983). CRCD rats were dosed at a TCDD level of 2,000 ng/kg bw/day for two weeks before mating experienced a significant increase ($p<0.05$, 19.5%) in pre-implantation loss, while doses of 500 and 2,000 ng/kg bw/day produced significant increases in post-implantation loss, at 10.2% and 30.3%, respectively. Significant fetal weight reduction was also observed at the 2,000 ng/kg bw/day dose.

The number of pups and survival rates of newborns have been reported to decrease as a result of treating dams with TCDD. Murray *et al*. (1979) treated Sprague Dawley rats with diets of 0, 1, 10 or 100 ng/kg bw/day for 90 days. The dosage period began at 7 weeks and ran for 90 days, at which time the F0 rats were mated to produce the F1a generation. The F0 rats were then mated again 33 days later to produce the F1b generation. The F1a and F1b rats were mated at an average age of 130 days to produce the F2 and F3 generations, respectively. At 100 ng/kg bw/day, the F1a and F1b generations were smaller in number ($p<0.05$) and F1a pups were all stillborn. At 10 ng/kg bw/day, the F2 and F3 generations were smaller in number ($p<0.05$) and had significantly lower survival rates to 21 days, 86% and 83%, compared to 90% for controls.

Holtzman rats receiving a single oral dose of TCDD of 1,000 ng/kg bw/day on day 15 of gestation (Bjerke and Peterson 1994; Mably *et al*. 1992) experienced a significant reduction in body weight of offspring at birth and at 5 days postpartum. Offspring survival rates were significantly reduced as well, with Bjerke and Peterson (1994) reporting a 30% decline and Mably *et al*. (1992) reporting a 15% decline.

Bjerke *et al*. (1994) dosed Holtzman rats with 700 ng/kg bw TCDD on day 15 of gestation by gavage and observed survival and body weights to 7 days. This study included pup exposure both *in utero* and through lactation. Pup mortality was significantly higher in test animals than in controls (23% vs 7%) and mean body weight of male offspring was decreased by 89-92% of control between birth and 7 days post partum.

Gray and Ostby (1995) administered 1,000 ng/kg bw of TCDD by gavage in Long-Evans hooded rats on gestation day 8 and observed significantly reduced fertility of the female offspring compared to controls. Breeding of female offspring was

monitored for 140 days starting at 233 days of age and only 19% produced a fifth litter compared to 61% for the control. Holtzman rats given a single oral dose of 1,000 ng/kg bw TCDD on gestation day 8 had neonatal viability to post-natal day 16 reduced by 50% in the treatment group, compared to 2.5% in controls.

Doses of 2,3,4,7,8-pentachlorodibenzofuran were observed to cause fetal mortality in rats. Couture *et al*. (1989) treated Fisher 344 rats with a dose of 2,3,4,7,8-pentachlorodibenzofuran ranging from 0 to 150,000 ng TEQ/kg bw/day on days 8, 10 or 12 of gestation with the animals being sacrificed on day 20. Rats dosed with 50,000 ng TEQ/kg bw on day 8 of gestation had significantly higher fetal mortality (9.93%) compared to controls (2.78%). The dose of 150,000 ng TEQ/kg bw administered on days 10 or 12 of gestation also produced significantly higher fetal mortality (91.7% and 81.1%, respectively) compared to controls (0% and 7.86%). Fetal weight was correlated with dose and fetal toxicity, and teratogenic effects were observed at the highest dose for administration on all three days, though significantly on days 8 and 12 only.

Rats and mice treated with 3,3',4,4'-tetrachlorobiphenyl (PCB 77) suffered embryotoxic effects to the fetuses including death and resorption (Marks *et al*. 1989; d'Argy *et al*. 1987; Wardell *et al*. 1982). Wardell *et al*. (1982) observed significant embryonic mortality (14% resorption) in Sprague Dawley rats when dams were exposed to a dose of 300 ng TEQ/kg bw/day of PCB77 on days 6-18 of gestation. d'Argy *et al*. (1987) reported a significant number of resorptions (37%) when C57BL/6 (B6) mice were administered an oral dose of 2,500 ng TEQ/kg bw/day of 3,3',4,4'-pentachlorobiphenyl (PCB 77) on day 11 of gestation.

Marks *et al*. (1989) reported a PCB 77 dose-related increase of the percentage of implants resorbed by mice administered 400 (7%) to 6,400 (82.5%) ng TEQ/kg

bw/day; a significant increase was determined at 1,600 (16.4%) ng TEQ/kg bw/day. In addition, the average number of live fetuses per dam was significantly reduced (21.6%) at 1,600 ng TEQ/kg bw/day and above (Marks *et al*. 1989). Rands *et al*. (1982) observed that pregnant rats dosed at 300 ng TEQ/kg bw/day of PCB 77 on day 6 to 18 of gestation experienced a statistically significant increase in mortality in offspring having a gestation period of 21 days or more. The results showed a trend toward decreased viability with increasing gestational time; this was also observed by Linzey (1987).

Heaton *et al*. (1995) investigated the reproductive effects of dietary exposure to planar halogenated hydrocarbons (PCDDs, PCDFs, PCBs, and TEQs) for adult mink over a 182 day period. The study began prior to mating and exposure continued after the kits were whelped. In this study, carp were collected from contaminated and reference sites near Saginaw Bay, MI. Their TEQ body burden was measured and the fish were incorporated into mink feed at levels of 0, 10, 20, and 40% contaminated carp. This resulted in TEQ dietary concentrations of 1.03, 19.4, 40.0, and 80.8 ng TEQ/kg feed and translated to daily doses of 0.25, 3.60, 6.80, and 10.7 ng TEQ/kg bw/d. All of the treatment levels affected reproduction in mink. The lowest dose significantly reduced kit body weight at three and six weeks to 67 and 79% of control body weights, respectively. At the highest dose level, the gestation length was reduced to 91% of controls, average litter size was reduced to 3.3 compared to 5.7 for controls, and the number of kits born alive per female was reduced from 5.0 for controls to 0.7 for mink fed the diet containing 40% Saginaw Bay carp. The NOAEL and LOAEL were calculated to be 0.27 and 4.23 ng TEQs/mink/day. Survivability of kits to six weeks was reduced from 85% in control animals to 28% and 11% in the 10% and 20% carp diets, respectively. No kits survived to 6 weeks in the 40% carp dietary treatment.

Field Surveys

No field surveys or studies were found in the literature relating effects of TEQs to omnivorous mammals.

Effects Metrics

The effects metrics for TCDD-TEQs are based on the combined investigations conducted by Khera and Ruddick (1973) and Sparschu *et al*. (1971). These investigators both treated pregnant female rats (Wistar and Sprague-Dawley, respectively) with oral doses of TCDD (8 and 5 treatment levels, respectively) on days 6 to 15 (inclusive) of gestation. The authors reported a number of reproductive endpoints, including the number of live fetuses per female. Generally, there were no significant impacts on the number of live fetuses per female at doses under 1,000 ng TEQ/kg bw/d, but effects rose sharply to 100% mortality as the dose exceeded 4,000 ng TEQ/kg bw/d.

We fitted the dose-response data from the above two studies to a generalized linear model (GLiM) using SAS® (SAS Institute, Cary, NC). Fecundity data were subjected to a log-link function and a Poisson error distribution was assumed (Bailer and Oris 1997). The model parameters were $\beta_0 = 2.49$, $\beta_1 = -0.000677$, $se\beta_0 = 0.107$, $se\beta_1 = 0.000147$, and $corr\beta_0\beta_1 = -0.505$. The resulting dose-response curve for the reproductive effects of TCDD to rats produced an adequate model to fit the data ($F = 88.6$; $p < 0.0001$) and is shown in Figure I1-5.

2.2.2.3 Selenium

Selenium is an essential element in human and animal nutrition and is efficiently concentrated in living tissues. Absorption of oral radioselenite by rats is as high as 95 to 100% (Eisler 1985). Marine fish have tissue residues of approximately 2 mg/kg ww, a concentration 50,000 times that of the surrounding seawater (Wilber 1980). Though essential to life and naturally accumulated, excess selenium exposure has been associated with lethality, neurological, developmental, and reproductive effects (ATSDR 1996). The selenium compounds shown to be the most toxic to mammals by ingestion appear to be sodium selenite and sodium selenate (Olson 1986). Both sodium selenate and sodium selenite are used as livestock feed supplements to prevent selenium deficiency diseases and both have been detected at chemical waste sites (NTP 1994).

Survival

Some of the earliest toxicological work done with selenium was conducted by Franke and Moxon (1936). These investigators established the median lethal intraperitoneal dose of sodium selenate to rats at 5.25 to 5.75 mg/kg bw and sodium selenite at 3.25 to 3.5 mg/kg bw. Other forms of selenium were shown to be less toxic by other researchers: diselenodipropionic acid with a reported LD_{50} of 25 mg/kg bw (Moxon *et al*. 1938); trimethylselenonium LD_{50} at 49.4 mg/kg bw (Obermeyer *et al*. 1971); dimethyl selenide has an LD_{50} of 1,600 mg/kg bw (McConnell and Portman 1952); and elemental selenium has an LD_{50} of 6,700 mg/kg bw (Cummins and Kimura 1971). Pletnikova (1970) examined the oral route of exposure and established a single oral dose LD_{50} for sodium selenite to the white mouse to be 7.75 mg/kg bw. The albino rat was also tested and had an oral LD_{50} of 10.5 mg/kg bw. Smith and Westfall (1937) claimed that the route of administration was not an important factor in

selenium toxicity due to the rapid and complete absorption of soluble selenium compounds.

Adult female CD-1 rats were administered sodium selenite by gavage for 8 days at levels of 2.5, 5, 10, 20, and 40 mg/kg in a study by Plasterer *et al*. (1985). Mice were treated in groups of ten and all were 61 to 71 days old at the initiation of the experiment. There was no evidence of a weight change in the animals and mortality was observed in each of the treatment levels. The LD_{50} was established, using probit analysis, at 8.4 mg/kg bw/d with a 95% confidence limit of 6.0 - 12.0 mg/kg bw/d. The 8 day LD_{10} was determined to be 7.0 mg/kg bw/d.

The effects of sodium selenite were also investigated with respect to the short- and long-term survival of Sprague-Dawley rats (Jacobs and Forst 1981). Five groups of five females were provided with water *ad libitum* treated with sodium selenite at concentrations of 1, 4, 8, 16, and 64 mg/L Se. Using an estimated body weight of 204 g and daily water intake of 31 mL (TERA 2002), the drinking water concentrations translate to daily doses of 0.15, 0.61, 1.22, 2.44, and 9.73 mg/kg bw/d for young female rats. For the 35 week exposure, rats were started at 5 or 12 weeks of age and monitored for growth and survival until death or the conclusion of the experiment. The group started at 5 weeks experienced significant mortality starting at 16 mg/L and complete mortality at the 64 mg/L treatment level. Animals that were started on the treatment at 12 weeks of age experienced significant mortality only at the highest treatment level, with all animals dying within 18 days. Growth was also measured for these two groups, with weight gain exhibiting a negative correlation with treatment level. In both groups, males and female rats both lost weight over the course of the experiment while controls and lower treatment levels gained weight. Longer studies involving similar animals, procedures, and measurement endpoints were conducted with exposure periods of 61 and 116 weeks to 4 mg/L selenium in

drinking water (Jacobs and Forst 1981). The longer exposures elicited no adverse effects to rats for survival or reproduction.

Schroeder and Mitchener (1971a) exposed weanling Long-Evans rats to sodium selenate or sodium selenite in drinking water for 1 year at 0 or 2 mg selenium/litre. After 1 year, the selenium selenate treatment concentration was increased to 3 mg/L. The group given the sodium selenate performed as well as controls, both reaching 90% mortality at approximately 1100 days. The male rats given the sodium selenite drinking water solution reached 50% mortality in 58 days, and females in 342 days. There was also a significant lag in body weight gain in males and female rats up until their deaths. The drinking water concentration of 2 mg/L was estimated to translate to a daily dose of 0.28 mg Se/kg bw/d (ATSDR 1996). Rosenfeld and Beath (1954) provided drinking water containing potassium selenate to rats at a concentration such that a daily dose of 1.05 mg Se/kg bw/d was achieved. The exposure period was 8 months and no mortalities were observed.

A study commissioned by the National Toxicology Program (NTP 1994) investigated the effects of sodium selenate given to rats and mice in drinking water over 13 weeks. Animals were divided into single sex groups of 10 and given drinking water treated with levels of 0, 3.75, 7.5, 15, 30, or 60 mg/L sodium selenate. At the conclusion of the study, the surviving animals were sacrificed and all were examined for hematology, clinical chemistry, urinalysis (rats only), histopathology, and reproductive system effects. The treatment concentrations were estimated to deliver daily doses of 0, 0.1, 0.2, 0.4, 0.6, 1.1 (males), or 0.8 (females) mg selenium/kg bw/d for rats and 0, 0.3, 0.5, 0.8, 1.5, or 2.6 mg/kg bw/d selenium for mice (ATSDR 1996). All male and female rats treated at the 60 mg/L level died within 11 and 6 weeks, respectively, while the mice were not affected at any of the concentrations. Growth of male and female mice and rats was reduced in the 30 and 60 mg/L treatments. The

sodium selenate treatments were associated with increased incidences of renal papillary regeneration in rats starting at water concentrations of 7.5 mg/L. This may have been due to dehydration as water consumption also decreased with increasing selenium concentration. No lesions related to sodium selenate administration occurred in mice.

The National Toxicology Program (NTP 1994) conducted a similar study using sodium selenite. The same 13 week drinking water protocol was used, however, for this experiment the treatment levels were reduced to 0, 2, 4, 8, 16, or 32 mg/L. These concentrations were estimated to deliver daily doses of 0, 0.08, 0.13, 0.2, 0.4, 0.8 (males), or 0.9 (females) mg/kg bw/d selenium for rats and 0, 0.14, 0.3, 0.5, 0.9, or 1.6 mg/kg bw/d selenium for mice. The only mortality in this study was in the highest treatment group of female rats. Two died in this group and all other animals survived to the conclusion of the study. Weight loss was also experienced by rats and mice over the course of the study as body weights of those in the highest treatment group were reduced by 17 and 54%, respectively.

Reproduction

Acute and subacute exposures to selenium in feed and drinking water does not appear to affect the fertility of female animals unless the intake is sufficiently high to cause general toxicity. In instances where the treatment levels are sufficiently high, general toxicity precludes any specific reproductive effects (Barlow and Sullivan 1982; Nobunaga *et al*. 1979). Chronic exposures have been shown to reduce fertility and to reduce the viability of the offspring of pairs that are able to conceive at doses somewhat below short term toxicity thresholds (Schroeder and Mitchener 1971b; Wahlstrom and Olson 1959; ATSDR 1996).

In a study conducted by Parshad (1999), albino Wistar rats were given daily intraperitoneal injections of sodium selenite at 2.0 or 4.0 mg/kg bw/d for 30 days. Animals in these groups experienced 14 and 40% mortality, respectively, over the course of the experiment and neither treatment had an effect on the length of the first two oestrus cycles. Examination of the ovaries at the conclusion of the experiment indicated that 21% of females in the low dose group and 60% of females in the high dose group had cystic follicles. Surviving animals with no cysts showed no signs of corpora lutea, indicating non-functional ovaries. In a subsequent study, the same investigators treated rats with intraperitoneal injections of sodium selenite at 2 and 4 mg/kg bw/d for the 4 days of the oestrus cycle and then mated the females with fertile males. This procedure resulted in 12 and 28% mortality, respectively, and surviving females were sacrificed for examination on day 14 of gestation. The percent of females that conceived was reduced from 92% in controls to 73% in the low dose group and 50% in the high dose group. Significant reductions were also observed in number of corpora lutea per female, the number of live embryos per litter and the number of implantation sites per litter.

Parshad and Sud (1989) have demonstrated that selenium is a reproductive toxicant to male rats. In their study, male Wistar rats were fed wheat grains that had naturally accumulated selenium to average levels of 12.5 mg/kg for 4 weeks. Rats in the treatment group were expected to have decreased food consumption and body weight gain and so a third group of animals was underfed to compare results. As expected, the treatment and underfed groups were both undersized compared to controls and had significantly lower testis weights. There were no spermatozoa in the lumen of the seminiferous tubules in rats fed the wheat with naturally accumulated selenium.

Schroeder and Mitchener (1971b) conducted a three generation study in which CD mice were exposed to 3 mg/L sodium selenate in drinking water and 0.056 mg/kg in

feed (estimated to be 0.76 mg selenium/kg bw/day; Sample *et al*. 1996). Five pairs of mice or rats were randomly selected for the first generation and subsequent generations were comprised of five pairs of the progeny of the previous generation. Control mice were bred for four generations with an average of 10-11 pups per litter. By the third generation of mice maintained on the selenium laced drinking water, only three litters were produced with an average of 7.6 pups per litter, compared with third generation controls which produced 22 litters with an average of 10.5 pups per litter. The total number of pups declined from 197 in the first generation to 169 in the second and 23 in the third. The number of runts per generation increased from 18% to 24% to 70% in the third generation, compared to less than 1% in all three generations of control animals.

Plasterer *et al*. (1985) treated female CD1 mice with sodium selenite at 7.0 mg/kg bw/d for 8 days. The dose level was selected as being just below the threshold of adult lethality and was administered on days 7-14 of gestation. Mice in this experiment showed no significant signs of reproductive toxicity for either total number of pregnant females, total number that delivered, or reproductive index.

Field Surveys

Selenium poisoning is a hazard to livestock in areas naturally rich in selenium (ATSDR 1996; Wilbur 1980; Rosenfeld and Beath 1964). "Blind staggers" is a condition symptomatic of cattle and sheep grazing in such areas. Animals wander from the herd as their vision fails and, as the poisoning continues, their behavior becomes more erratic, limbs become weak, and the animals finally succumb to respiratory failure (Wilbur 1980). This effect in livestock is typically associated with Se concentrations of 400 to 800 mg/kg in plant material (Eisler 1985). Chronic exposures to selenium can lead to "alkali disease" which is characterized by growth retardation, inhibition of reproduction, hair loss, abnormal hoof formation, erosion

of the cartilages, and degeneration of heart, kidney, and liver. (Wilbur 1980). It has been postulated that Se displaces sulfur in keratin, resulting in structural changes in hair, nails, and hooves (Eisler 1985). Alkali disease is associated with the consumption of grains containing 5 - 40 mg selenium/kg over weeks or months (WHO 1987).

Due to concerns of atmospheric metal deposition and acid rain increasing metal mobilization, aquatic-dependent mammals were sampled from remote lakes in the Canadian Precambrian Shield (Wren 1984) for several metals. Selenium concentrations in otter and raccoon muscle tissue averaged approximately 0.2 mg/kg ww while concentrations in liver and kidney averaged 2-3 mg/kg ww. These tissue concentrations are approaching the criteria proposed by Eisler (2000a) of 3-6 mg/kg for kidney and 12-15 mg/kg for liver as thresholds for protection against selenium toxicity.

The Kesterson National Wildlife Refuge in California suffered unusually high rates of embryonic mortality and abnormalities in the young of nesting aquatic birds in the early 1980s (Clark 1987). Tissue and media analysis revealed that high selenium concentrations may have contributed to the effects and irrigation drain water was identified as a source of the contamination. Clark (1987) sampled a number of small mammals in the Kesterson area and nearby reference areas and found no adverse effects of selenium on wild mammals. Selenium concentrations in various mammal liver tissues ranged from a maximum of 250 mg/kg dw in the California vole to 0.91 mg/kg dw in the long-tailed weasel. The highest mammalian liver tissue concentration in the reference areas was 3.7 mg/kg dw in the house mouse. Clark *et al*. (1989) examined raccoons in the Kesterson Refuge and found liver tissue selenium concentrations of 19.9 mg/kg dw, but no evidence of selenium toxicity in any of a number of measurement endpoints. Rhian and Moxon (1943) noted inhibited growth

in dogs associated with liver concentrations of 16-67 mg/kg dw and Rosenfeld and Beath (1964) reported that 4-32 mg/kg dw in livestock liver tissue was associated with "blind staggers" disease. Despite raccoons and voles surpassing these concentrations in the Kesterson Refuge, no pathologies were found in the animals.

Effects Metrics

Selenium toxicity is expressed over a narrow dose range with lethal and sublethal effects sharing the same dose range (ATSDR 1996). Studies in which reproductive effects were investigated found that for exposures in the range of one month, the fertility of female animals was unaffected, unless the intake was high enough to cause general toxicity (Barlow and Sullivan 1982; Nobunaga *et al*. 1979). Mortality is a more sensitive endpoint. The effects metrics for selenium are, therefore, based on the investigation conducted by Jacobs and Forst (1981) in which 5-week old female Sprague-Dawley rats were given water *ad libitum* treated with 1, 4, 8, 16, and 64 mg/L sodium selenite for 35 days. To convert these drinking water concentrations to daily doses, we multiplied the drinking water concentration by the daily water intake rate of 0.152 L/kg bw/d (TERA 2002). The estimated daily doses were 0.15, 0.61, 1.22, 2.43, and 9.73 mg/kg bw/d. Animals in the three lowest treatment groups suffered no significant mortalities, while the groups receiving the two highest doses suffered 80 and 100% mortality respectively.

We fitted the dose-response data from the above two studies to a generalized linear model (GLiM) using SAS® (SAS Institute, Cary, NC). Mortality data were subjected to a probit-link function and a binomial error distribution was assumed (Bailer and Oris 1997). The model parameters were $\beta_0 = 3.08$, $\beta_1 = -1.62$, se$\beta_0 = 1.05$, se $\beta_1 = 0.582$, and corr$\beta_0\beta_1 = -0.910$. The resulting dose-response curve for the reproductive effects of sodium selenite to rats produced an adequate model to fit the data ($F = 48067.1$; $p < 0.0001$) and is shown in Figure I1-6.

2.2.2.4 Total PCBs

The potential effects of PCBs to omnivorous mammals include mortality (Green *et al*. 1975; Kimbrough *et al*. 1972), reduced litter size and survival at birth (Golub *et al*. 1991; Spencer 1982), decreases in body and organ weight (Kimbrough *et al*. 1972; Komives 1979), and others (Safe 1994; French *et al*. 2001). This section focuses on long term exposures and effects to survival, growth, and reproduction of omnivorous mammals.

Mortality

Linder *et al*. (1974) treated 3 to 4 week old Sherman-strain male rats with single oral doses of Aroclors 1254 and 1260. Single-dose oral LD_{50} s from this investigation were 1,295 and 1,315 mg/kg bw/d, respectively. The toxicity of a single dose of Aroclor 1254 to Wistar male and female rats at 30, 60 and 120 days of age was investigated by Grant and Phillips (1974). The LD_{50} s ranged from 1,300 to 2,500 mg/kg bw/d.

Green *et al*. (1975) dosed Osborne-Mendel male rats with Aroclor 1254 by oral intubation over an exposure period of 5 days. The investigators observed that a dose of 300 mg/kg bw/d caused 50% mortality in these animals at the conclusion of the exposure period. These animals also experienced a mean weight loss of 0.056 kg during the exposure period. Female Sherman-strain rats treated with doses of Aroclor 1260 of 7.2, 38.2 and 72.4 mg/kg bw/d for eight months experienced 10, 20, and 80% mortality, respectively. No mortality was observed in controls and male rats.

Hornshaw *et al*. (1986) conducted a 28 day study of acute oral toxicity of Aroclor 1254 to mink. Investigators conducting this study noted a decrease in both feed consumption and body weight over the 28 days. Mink fed a diet containing nominal

concentrations of Aroclor 1254 of 58.3 mg/kg experienced 50% mortality compared to all lower treatment levels, which showed no mortality.

Reproduction

Sprague-Dawley rats were investigated for the reproductive effects of PCB exposure during gestation (Spencer 1982). Dams were maintained on 8 treatment level diets containing Aroclor 1254 on days 6 through 15 of gestation. Fetal birth weight was reduced by 11% and fetal survival was reduced by 28% after dams received oral doses of 7.47 and 17.05 mg/kg bw/d, respectively. Overmann *et al*. (1987) treated female Wistar rats from conception to weaning with dietary doses of Aroclor 1254. Pups whose mothers were exposed to Aroclor 1254 at 2.83 mg/kg bw/d were significantly lighter compared to controls on days 14 and 21. Brezner *et al*. (1984) and Sager and Girard (1994) also observed significantly reduced weight gain in pups in Aroclor 1254 treatment groups compared to controls. Merson and Kirkpatrick (1976) fed Aroclor 1254 to pairs of white-footed mice at a dietary dose of 30.9 mg/kg bw/d for 60 days during which time litters were produced. The controls had a litter production rate of 84.6% while the PCB-treated group had a litter production rate of only 29.6%. No offspring of the PCB-treated group survived beyond 21 days

Linder *et al*. (1974) conducted a two-generation study in which groups of 20 female and 10 male Sherman rats were exposed to diets of Aroclor 1254 at doses of 0.06, 0.32, 1.5, and 7.6 mg/kg bw/d. Exposure times ranged from 62 to 274 days. The F0 rats began the treatments at 3 to 4 weeks of age and continued through mating, gestation and lactation until the rats were euthanized. The F0 rats were mated when they reached 3 and 7 months old to produce the F1a and F1b generations, respectively. The F1b rats were selected at weaning for mating when 3 months old to produce the F2a generation. The F1b rats were mated a second time when 8 months old to produce the F2b generation. The 7.6 mg/kg bw/d Aroclor 1254

treatment level caused significantly reduced litter sizes (13.7%) in the F1a generation. The 1.5 mg/kg bw/d treatment level caused significantly reduced litter sizes in the F1b (15.4%), F2a (15.2%) and F2b (24.4%) generations. No effects on litter size were found in either generation of rats fed 0.32 mg/kg bw/d or 0.06 mg/kg bw/d of Aroclor 1254. Although reproduction was not affected at lower dietary levels of PCBs, a significant increase in liver weight in 21-day-old weanlings was found at all levels of exposure.

White-footed mice (*Peromyscus leucopus*) exposed to a chronic dose of 1.55 mg/kg bw/d of Aroclor 1254, starting at 12 weeks of age, experienced impairment of reproductive success. PCB-exposed laboratory mice had statistically significant longer intervals between births, smaller litter sizes at birth (25%, averaging one less individual), and smaller litter sizes at weaning (control = 98% survival; PCB treatment = 56% survival; Linzey 1987). The surviving offspring from this study were used to investigate the effects of PCBs on second generation mice (Linzey 1988). The reproductive success of the second-generation PCB-treated mice was reduced compared to the parental generation. For example, 42% of the PCB-treated pairs in the parental generation produced litters while only 4% of the PCB-treated pairs in the second generation produced litters. In addition, only 47% of the second-generation mice survived to weaning compared to 56% in the parental generation. The second-generation PCB-treated mice exhibited poor growth of reproductive organs, the females being most affected. The data in the second-generation study were too few to test for statistical significance. However, these results suggest that effects of chronic exposure to PCBs are cumulative through generations.

McCoy *et al*. (1995) maintained oldfield mice over three generations (P1, F1 and F2 generations) on a dose of Aroclor 1254 of 0.678 mg/kg bw/d to investigate effects on fertility, growth and survival. In the PCB-exposed first generation, the mean birth

weight of pups was significantly lower (-13%) than that of the P1 non-exposed control group. Weaning weight was also significantly lower in PCB-exposed first generation animals (-13%) compared to the P1 non-exposed group. Notable differences between PCB-exposed and control mice also occurred in the second generation mice, including lower mean birth weight (-14.8%) and lower weaning weight (-15%). The percentage survival to weaning was 23% for the second generation, compared to 55% for the first generation.

Linder *et al*. (1974) treated groups of 20 female and 10 male Sherman rats to diets of Aroclor 1260 at dose levels of 0.39, 1.5 and 7.4 mg/kg bw/d. In this two-generation reproduction study, maternal exposure times ranged from 68 to 188 days. No effects on litter size were found in either first or second generation of rats fed >0.39 mg/kg bw/d of Aroclor 1260. An increase in liver-to-body weight ratios was observed at all dietary levels in F1b males between 5 and 7 months old, but only at 7.4 mg/kg bw/d in the F1b females. In the same study, there was no significant difference from controls for number of litters born and survival at weaning. In a one-generation reproduction study using a higher dose of 35.4 mg/kg bw/d, Linder *et al*. (1974) observed a significant decrease in litter size at birth (-31%) and survival to weaning (-73%).

Growth

Seven-week-old Sprague-Dawley rats given daily doses of 50 mg/kg bw/d Aroclor 1254 by oral intubation experienced significant decrease in body weight after 11 days of treatment and significant decrease in food intake after 4 days (Komives 1979). At 500 mg/kg bw/d, the effects occurred sooner. A significant decrease in body weight in male rats was also observed by Grant *et al*. (1974) fed a dose of 139 mg/kg bw/d of Aroclor 1254 for 60 days of exposure. After 246 days of exposure, a significant decrease in body weight was also reported at a dose of 5.79 mg/kg bw/d. Body

weight gain was inhibited in male and female Sprague-Dawley rats at a dose of 28.5 mg/kg bw/d and 8.2 mg/kg bw/d after a 39 day exposure (Kerkvliet and Kimeldorf 1977).

Other Effects

Orberg and Kihlstrom (1973) observed that a 10-week treatment of mice with Clophen 60 at 0.8 mg/kg bw/d caused prolonged estrous cycles. Brezner *et al*. (1984) noted a similar effect in rats maintained on 10 mg/kg bw/d Aroclor 1254 for 6 weeks. Decreased implantation and embryonic survival in rats are associated with a prolonged estrous cycle (Butcher and Pope 1979).

Liver weights were significantly different for rats exposed to Aroclor 1254 and 1260 (Kimbrough *et al*.1972) compared to controls. Similar effects have been reported for offspring of exposed individuals. PCB mixtures affected weight gain, liver weight, uterine weight during proestrus, delayed puberty, and impaired thyroid function in rats and mice including offspring of rats (Linzey 1987; Collins and Capen 1980; Grant and Phillips 1974; Pantaleoni *et al.* 1988; Linder *et al.* 1974; Overmann *et al.* 1987; Storm *et al.* 1981; Kerkvliet and Kimeldorf 1977; Sanders and Kirkpatrick 1977; Kimbrough *et al.* 1972; French *et al.* 2001; Kasza *et al.* 1978; Talcott and Koller 1983; Sager and Girard 1994).

Voltura and French (2000) conducted a study on white-footed mice, in which the energetic cost of exposure to PCBs was determined. Exposure to harmful substances is likely to be metabolically expensive and may result in a trade-off between energy spent to detoxify and excrete contaminants and energy allocated to growth and reproduction. Four-month-old mice were fed diets for six weeks containing a mixture of 2:1 Aroclor 1242:1254 at levels ranging from 0 to 5.4 mg/kg bw/d. The diets were continued for 10 months. At six weeks of exposure, there were no differences in food

intake or body mass. After one year, compared to the control group, mice fed the highest dose tended to be heavier, and had higher food intake and oxygen consumption. These results suggested that there is an energetic cost to long-term exposure that may influence energy acquisition and allocation.

Field Surveys

No field surveys or studies were found in the literature involving the exposure of omnivorous mammals to total PCBs.

Effects Metrics

The effects of total PCBs to omnivorous mammals in the Calcasieu Estuary is ideally done using the results of controlled investigations that treated a relevant omnivorous mammal with at least five dose levels and measured for effects on growth, survival, or reproduction. Controlled toxicity studies on these endpoints were not available for PCBs. As a result, surrogate mammals for these representative species were used to assess toxicity. For omnivorous mammals, the rat was used as a surrogate mammal, when data were available.

The Spencer (1982) study used enough treatment levels to generate a dose-response curve for the effects of total PCBs to the reproduction of rats. Female rats were administered dietary doses of Aroclor 1254 at 8 treatment levels on days 6 through 15 of gestation and mortality of pups at birth was monitored. The curve fitting of dose-response data to a generalized linear model (GLiM) was performed using SAS® (SAS Institute, Cary, NC). Mortality data required a logit link function and a binomial error distribution was assumed (Bailer and Oris 1997). Figure I1-7 presents the dose-response curve for mortality at birth of rats. In this analysis, Abbott's formula was used to correct for control mortality (Newman 1995). Because mean responses were used in the analysis (the raw data were not available), fiducial limits

and goodness of fit were not estimated. The model parameters were β_0 = -15.3804, β_1 = 3.7136, seβ_0 = 1.1897, seβ_1 = 0.2865, and corr$\beta_0\beta_1$ = -0.9971. The dose-response model was significant at $p<0.0007$ (F value = 30.75, 6 degrees of freedom).

2.2.3 Risk Characterization

In the risk characterization phase of the probabilistic risk assessment, the results of the exposure assessment (i.e., reverse cumulative distribution functions) and effects assessment (i.e., dose-response relationships) were integrated to develop risk curves for each COC and each AOC. Ideally, risk characterization involves three major lines of evidence: comparison of modeled exposure to lab-derived effects metrics, *in situ* or whole media toxicity tests, and biological surveys. For omnivorous mammals, however, the latter two lines of evidence are not available. We therefore rely on the risk curves generated from the comparison of modeled exposure to laboratory derived dose-response curves.

3.0 Results

3.1 Probabilistic Exposure Assessment

Mercury in Bayou d'Inde AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to total mercury in the Bayou d'Inde AOC could range from a minimum of 0.00258 to a maximum of 0.0219 mg/kg bw/day. The mean exposure

is 0.00720 mg/kg bw/day and the median exposure is 0.00692 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00436 and 0.0109 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.00629 to a maximum of 0.0351 mg/kg bw/day. The mean exposure is 0.0146 mg/kg bw/day and the median exposure is 0.0143 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00979 and 0.0209 mg/kg bw/day. Figure I1-8 and Figure I1-9 depict the cumulative distributions of total mercury intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.62) followed by gross energy of fish (r_p = -0.54). The most important variable for the hypothetical small receptor was the gross energy of fish (r_p = -0.65), followed by the free metabolic rate slope term (r_p = 0.54).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Bayou d'Inde AOC are shown in Figure I1-8. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00249 and 0.0102 mg/kg bw/day. The 50th percentile ranges between 0.00409 and 0.0145 mg/kg bw/day, and the 90th percentile ranges between 0.00544 and 0.0243 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.00482, the 50th percentile is 0.00692, and the 90th percentile is 0.00986 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-9. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00562 and 0.0222 mg/kg bw/day. The 50th percentile ranges between 0.00868 and 0.0305 mg/kg bw/day, and the 90th percentile ranges between 0.0111 and 0.0476 mg/kg

bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0105, the 50th percentile is 0.0143, and the 90th percentile is 0.0191 mg/kg bw/day.

Mercury in the Reference Areas

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to total mercury in the Calcasieu Estuary reference areas could range from a minimum of 0.000369 to a maximum of 0.00312 mg/kg bw/day. The mean exposure is 0.00103 mg/kg bw/day and the median exposure is 0.000991 mg/kg bw/day. Ninety percent of exposure estimates are between 0.000631 and 0.00154 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.000899 to a maximum of 0.00497 mg/kg bw/day. The mean exposure is 0.00209 mg/kg bw/day and the median exposure is 0.00204 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00141 and 0.00294 mg/kg bw/day. Figure I1-10 and Figure I1-11 depict the cumulative distributions of total mercury intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.64) followed by gross energy of fish (r_p = -0.49). The most important variable for the hypothetical small receptor was the gross energy of fish (r_p = -0.61), followed by the free metabolic slope term (r_p = 0.57).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Calcasieu Estuary reference areas are shown in Figure I1-10. The 10th percentile of the probability envelope formed by the lower and upper bounds

ranges between 0.000381 and 0.00190 mg/kg bw/day. The 50th percentile ranges between 0.000640 and 0.00273 mg/kg bw/day, and the 90th percentile ranges between 0.000916 and 0.00458 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.000699, the 50th percentile is 0.000991, and the 90th percentile is 0.00140 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-11. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.000857 and 0.00377 mg/kg bw/day. The 50th percentile ranges between 0.00137 and 0.00536 mg/kg bw/day, and the 90th percentile ranges between 0.00190 and 0.00859 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.00153, the 50th percentile is 0.00204, and the 90th percentile is 0.00270 mg/kg bw/day.

TCDD-TEQs in Bayou d'Inde AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to TCDD-TEQs in the Bayou d'Inde AOC could range from a minimum of 0.477 to a maximum of 16.7 ng/kg bw/day. The mean exposure is 1.76 ng/kg bw/day and the median exposure is 1.56 ng/kg bw/day. Ninety percent of exposure estimates are between 0.854 and 3.31 ng/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 1.06 to a maximum of 29.7 ng/kg bw/day. The mean exposure is 3.58 ng/kg bw/day and the median exposure is 3.21 ng/kg bw/day. Ninety percent of exposure estimates are between 1.88 and 6.58 ng/kg bw/day. Figure I1-12 and Figure I1-13 depict the cumulative distributions of TCDD-TEQs intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the concentration of TEQs in invertebrates was the most important variable for the hypothetical average-sized receptor (Pearson

correlation coefficient (r_p) = 0.71) followed by the free metabolic rate power term (r_p = 0.43). The most important variable for the hypothetical small receptor was the concentration of TEQs in invertebrates (r_p = 0.77), followed by the free metabolic rate slope term (r_p = 0.35).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Bayou d'Inde AOC are shown in Figure I1-12. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.188 and 7.83 ng/kg bw/day. The 50th percentile ranges between 0.317 and 12.1 ng/kg bw/day, and the 90th percentile ranges between 0.419 and 20.9 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.973, the 50th percentile is 1.56, and the 90th percentile is 2.76 ng/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-13. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.478 and 14.1 ng/kg bw/day. The 50th percentile ranges between 0.759 and 21.3 ng/kg bw/day, and the 90th percentile ranges between 0.969 and 34.4 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 2.10, the 50th percentile is 3.21, and the 90th percentile is 5.48 ng/kg bw/day.

TCDD-TEQs in Middle Calcasieu River AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to TCDD-TEQs in the Middle Calcasieu River AOC could range from a minimum of 0.280 to a maximum of 1.85 ng/kg bw/day. The mean exposure is 0.772 ng/kg bw/day and the median exposure is 0.744 ng/kg bw/day. Ninety percent of exposure estimates are between 0.474 and 1.16 ng/kg bw/day. For

hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.679 to a maximum of 3.24 ng/kg bw/day. The mean exposure is 1.57 ng/kg bw/day and the median exposure is 1.53 ng/kg bw/day. Ninety percent of exposure estimates are between 1.06 and 2.20 ng/kg bw/day. Figure I1-14 and Figure I1-15 depict the cumulative distributions of TCDD-TEQs intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.65) followed by the free metabolic rate slope term (r_p = 0.47). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.58), followed by the gross energy of fish (r_p = -0.56).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Middle Calcasieu River AOC are shown in Figure I1-14. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0574 and 2.99 ng/kg bw/day. The 50th percentile ranges between 0.0928 and 3.82 ng/kg bw/day, and the 90th percentile ranges between 0.119 and 6.28 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.524, the 50th percentile is 0.744, and the 90th percentile is 1.05 ng/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-15. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.143 and 5.46 ng/kg bw/day. The 50th percentile ranges between 0.2.18 and 6.78 ng/kg bw/day, and the 90th percentile ranges between 0.271 and 10.5 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 1.15, the 50th percentile is 1.53, and the 90th percentile is 2.04 ng/kg bw/day.

TCDD-TEQs in the Reference Areas

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to TCDD-TEQs in the reference areas of the Calcasieu Estuary could range from a minimum of 0.0742 to a maximum of 0.815 ng/kg bw/day. The mean exposure is 0.233 ng/kg bw/day and the median exposure is 0.223 ng/kg bw/day. Ninety percent of exposure estimates are between 0.135 and 0.364 ng/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.183 to a maximum of 1.29 ng/kg bw/day. The mean exposure is 0.474 ng/kg bw/day and the median exposure is 0.459 ng/kg bw/day. Ninety percent of exposure estimates are between 0.299 and 0.698 ng/kg bw/day. Figure I1-16 and Figure I1-17 depict the cumulative distributions of TCDD-TEQs intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the gross energy of fish was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = -0.63) followed by the free metabolic rate power term (r_p = 0.57). The most important variable for the hypothetical small receptor was the gross energy of fish (r_p = -0.74), followed by the free metabolic rate slope term (r_p = 0.49).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Calcasieu Estuary reference areas are shown in Figure I1-16. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0234 and 0.662 ng/kg bw/day. The 50th percentile ranges between 0.0416 and 0.892 ng/kg bw/day, and the 90th percentile ranges between 0.0549 and 1.65 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.152, the 50th percentile is 0.223, and the 90th percentile is 0.329 ng/kg bw/day.

The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-17. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0580 and 1.22 ng/kg bw/day. The 50th percentile ranges between 0.0964 and 1.63 ng/kg bw/day, and the 90th percentile ranges between 0.123 and 2.82 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.329, the 50th percentile is 0.459, and the 90th percentile is 0.637 ng/kg bw/day.

Selenium in Bayou d'Inde AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to selenium in the Bayou d'Inde AOC could range from a minimum of 0.0125 to a maximum of 0.0957 mg/kg bw/day. The mean exposure is 0.0346 mg/kg bw/day and the median exposure is 0.0334 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0216 and 0.0516 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.0301 to a maximum of 0.159 mg/kg bw/day. The mean exposure is 0.0703 mg/kg bw/day and the median exposure is 0.0687 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0482 and 0.0972 mg/kg bw/day. Figure I1-18 and Figure I1-19 depict the cumulative distributions of selenium intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.66) followed by the free metabolic rate slope term (r_p = 0.48). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.59), followed by the gross energy of invertebrates (r_p = -0.44).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in Bayou d'Inde AOC are shown in Figure I1-18. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0121 and 0.0555 mg/kg bw/day. The 50th percentile ranges between 0.0189 and 0.0802 mg/kg bw/day, and the 90th percentile ranges between 0.0232 and 0.129 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0237, the 50th percentile is 0.0334, and the 90th percentile is 0.0467 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-19. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0274 and 0.110 mg/kg bw/day. The 50th percentile ranges between 0.0408 and 0.157 mg/kg bw/day, and the 90th percentile ranges between 0.0491 and 0.240 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0521, the 50th percentile is 0.0687, and the 90th percentile is 0.905 mg/kg bw/day.

Selenium in Middle Calcasieu River AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to selenium in the Middle Calcasieu River AOC could range from a minimum of 0.0117 to a maximum of 0.0881 mg/kg bw/day. The mean exposure is 0.0328 mg/kg bw/day and the median exposure is 0.0317 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0204 and 0.0489 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.0281 to a maximum of 1.50 mg/kg bw/day. The mean exposure is 0.0666 mg/kg bw/day and the median exposure is 0.0651 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0457 and 0.0923 mg/kg bw/day. Figure I1-20 and

Figure I1-21 depict the cumulative distributions of selenium intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.66) followed by the free metabolic rate slope term (r_p = 0.48). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.59), followed by the gross energy of invertebrates (r_p = -0.51).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Middle Calcasieu River AOC are shown in Figure I1-20. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0110 and 0.0538 mg/kg bw/day. The 50th percentile ranges between 0.0175 and 0.0790 mg/kg bw/day, and the 90th percentile ranges between 0.0221 and 0.129 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0225, the 50th percentile is 0.0317, and the 90th percentile is 0.0442 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-21. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0248 and 0.107 mg/kg bw/day. The 50th percentile ranges between 0.0377 and 0.155 mg/kg bw/day, and the 90th percentile ranges between 0.0464 and 0.242 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0492, the 50th percentile is 0.0651, and the 90th percentile is 0.0857 mg/kg bw/day.

Selenium in Upper Calcasieu River AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to selenium in the Upper Calcasieu River AOC could range from a minimum of 0.0134 to a maximum of 0.0969 mg/kg bw/day. The mean exposure is 0.0373 mg/kg bw/day and the median exposure is 0.0361 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0232 and 0.0560 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.0323 to a maximum of 0.172 mg/kg bw/day. The mean exposure is 0.0759 mg/kg bw/day and the median exposure is 0.0740 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0521 and 0.106 mg/kg bw/day. Figure I1-22 and Figure I1-23 depict the cumulative distributions of selenium intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.66) followed by the free metabolic rate slope term (r_p = 0.48). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.59), followed by the gross energy of invertebrates (r_p = -0.51).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Upper Calcasieu River AOC are shown in Figure I1-22. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0129 and 0.0592 mg/kg bw/day. The 50th percentile ranges between 0.0203 and 0.0853 mg/kg bw/day, and the 90th percentile ranges between 0.0255 and 0.137 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0256, the 50th percentile is 0.0397, and the 90th percentile is 0.0619

mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-23. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0292 and 0.118 mg/kg bw/day. The 50th percentile ranges between 0.0438 and 0.167 mg/kg bw/day, and the 90th percentile ranges between 0.0533 and 0.256 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0561, the 50th percentile is 0.0740, and the 90th percentile is 0.0979 mg/kg bw/day.

Selenium in the Reference Areas

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to selenium in the reference areas of the Calcasieu Estuary could range from a minimum of 0.00936 to a maximum of 0.0657 mg/kg bw/day. The mean exposure is 0.0253 mg/kg bw/day and the median exposure is 0.0244 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0157 and 0.0380 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.0225 to a maximum of 0.117 mg/kg bw/day. The mean exposure is 0.0514 mg/kg bw/day and the median exposure is 0.0500 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0352 and 0.0720 mg/kg bw/day. Figure I1-24 and Figure I1-25 depict the cumulative distributions of selenium intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.65) followed by the free metabolic rate slope term (r_p = 0.48). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.58), followed by the gross energy of invertebrates (r_p = -0.55).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the reference areas of the Calcasieu Estuary are shown in Figure I1-24. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0114 and 0.0581 mg/kg bw/day. The 50th percentile ranges between 0.0185 and 0.0859 mg/kg bw/day, and the 90th percentile ranges between 0.0244 and 0.143 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0173, the 50th percentile is 0.0244, and the 90th percentile is 0.0343 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-25. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0258 and 0.115 mg/kg bw/day. The 50th percentile ranges between 0.0399 and 0.168 mg/kg bw/day, and the 90th percentile ranges between 0.0515 and 0.267 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.0379, the 50th percentile is 0.0500, and the 90th percentile is 0.664 mg/kg bw/day.

Total PCBs in Bayou d'Inde AOC

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to total PCBs in the Bayou d'Inde AOC could range from a minimum of 0.00224 to a maximum of 0.0190 mg/kg bw/day. The mean exposure is 0.00619 mg/kg bw/day and the median exposure is 0.00594 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00369 and 0.00947 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.00535 to a maximum of 0.0332 mg/kg bw/day. The mean exposure is 0.0126 mg/kg bw/day and the median exposure is 0.0122 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00815 and 0.0181 mg/kg bw/day. Figure I1-26 and

Figure I1-27 depict the cumulative distributions of total PCBs intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.60) followed by gross energy of fish (r_p = -0.52). The most important variable for the hypothetical small receptor was the gross energy of fish (r_p = -0.63), followed by the free metabolic rate slope term (r_p = 0.52).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Bayou d'Inde AOC are shown in Figure I1-26. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00166 and 0.00722 mg/kg bw/day. The 50th percentile ranges between 0.00273 and 0.0109 mg/kg bw/day, and the 90th percentile ranges between 0.00372 and 0.0180 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.00411, the 50th percentile is 0.00594, and the 90th percentile is 0.00855 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-27. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00374 and 0.0153 mg/kg bw/day. The 50th percentile ranges between 0.00583 and 0.0214 mg/kg bw/day, and the 90th percentile ranges between 0.00767 and 00337 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.00897, the 50th percentile is 0.0122, and the 90th percentile is 0.0166 mg/kg bw/day.

Total PCBs in the Reference Areas

Monte Carlo Analysis

The Monte Carlo analysis revealed that exposure of hypothetical average-sized omnivorous mammals to total PCBs in the Calcasieu Estuary reference areas could range from a minimum of 0.000400 to a maximum of 0.00351 mg/kg bw/day. The mean exposure is 0.00108 mg/kg bw/day and the median exposure is 0.00104 mg/kg bw/day. Ninety percent of exposure estimates are between 0.000654 and 0.00164 mg/kg bw/day. For hypothetical small omnivorous mammals, daily dose could range from a minimum of 0.000941 to a maximum of 0.00554 mg/kg bw/day. The mean exposure is 0.00220 mg/kg bw/day and the median exposure is 0.00214 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00147 and 0.00314 mg/kg bw/day. Figure I1-28 and Figure I1-29 depict the cumulative distributions of total PCBs intake rates for the hypothetical average-sized and small omnivorous mammals.

Sensitivity analysis indicated that the free metabolic rate power term was the most important variable for the hypothetical average-sized receptor (Pearson correlation coefficient (r_p) = 0.62) followed by the free metabolic rate slope term (r_p = 0.46). The most important variable for the hypothetical small receptor was the free metabolic rate slope term (r_p = 0.55), followed by the gross energy of fish (r_p = -0.53).

Probability Bounds Analysis

The probability bounds analysis estimated for hypothetical average-sized omnivorous mammals foraging in the Calcasieu Estuary reference areas are shown in Figure I1-28. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.000267 and 0.00203 mg/kg bw/day. The 50th percentile ranges between 0.000543 and 0.00304 mg/kg bw/day, and the 90th percentile ranges between 0.00107 and 0.00608 mg/kg bw/day. In comparison, the 10th percentile of the Monte

Carlo distribution is 0.000730, the 50th percentile is 0.00104, and the 90th percentile is 0.00148 mg/kg bw/day. The probability bounds estimated for hypothetical small omnivorous mammals are depicted in Figure I1-29. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.000592 and 0.00404 mg/kg bw/day. The 50th percentile ranges between 0.00116 and 0.00599 mg/kg bw/day, and the 90th percentile ranges between 0.00224 and 0.0116 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo distribution is 0.00159, the 50th percentile is 0.00214, and the 90th percentile is 0.00288 mg/kg bw/day.

3.2 Risk Assessment

For the AOCs in the Calcasieu Estuary, a low, indeterminate, and high category of risk was determined for omnivorous mammals. These categories of risk were derived using the following guidelines:

1. If the probability of exceeding 10% or greater effect is less than 20%, the risk to omnivorous mammals is considered low.
2. If the probability of exceeding 20% or greater effect is greater than 50%, the risk to omnivorous mammals is considered high.
3. All other outcomes are considered to have indeterminate risk.

Mercury in Bayou d'Inde AOC

Integration of the methylmercuric chloride effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to mercury in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental mercury contamination. The dose predicted to induce 1% effect is 0.0345 mg/kg bw/d, while exposure to mercury in the

Bayou d'Inde AOC is predicted to be less than 0.0128 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 3% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 78% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the methylmercuric chloride effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to mercury in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental mercury contamination. The dose predicted to induce 1% effect is 0.0345 mg/kg bw/d, while exposure to mercury in the Bayou d'Inde AOC is predicted to be less than 0.0245 mg/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have an 80% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/d. The lower and upper probability bounds of exposure have a 5% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Mercury in Reference Areas

Integration of the methylmercuric chloride effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to

mercury in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental mercury contamination. The dose predicted to induce 1% effect is 0.0345 mg/kg bw/d, while exposure to mercury in the Calcasieu reference areas is predicted to be less than 0.00180 mg/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 1% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of methylmercuric chloride effects curve with the Monte Carlo and probability bounds curves for the exposure of the small omnivorous mammals to mercury in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental mercury contamination. The dose predicted to induce 1% effect is 0.0345 mg/kg bw/d, while exposure to mercury in the Calcasieu reference areas is predicted to be less than 0.00344 mg/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 0% probability of total daily mercury intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 4% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

TCDD-TEQs in Bayou d'Inde AOC

Integration of the TCDD-TEQs effect curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to TCDD-TEQs in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental TCDD-TEQ contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to TCDD-TEQ in the Bayou d'Inde AOC is predicted to be less than 4.05 ng/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure have a 24% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the TCDD-TEQs effect curve with the Monte Carlo and probability bounds curves for the exposure of the small omnivorous mammals receptor to TCDD-TEQs in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental mercury contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to mercury in the Bayou d'Inde AOC is predicted to be less than 7.72 ng/kg bw/d 97% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure both have

100% probabilities of exceeding the Appendix G benchmark. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

TCDD-TEQs in Middle Calcasieu River AOC

Integration of the TCDD-TEQs effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to TCDD-TEQs in the Middle Calcasieu River AOC indicate that this receptor is at low risk for reproductive effects due to environmental TCDD-TEQ contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to TCDD-TEQ in the Middle Calcasieu River AOC is predicted to be less than 1.33 ng/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 99% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the TCDD-TEQs effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to TCDD-TEQs in the Middle Calcasieu River AOC indicate that this receptor is at low risk for reproductive effects due to environmental TCDD-TEQ contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to TCDD-TEQ in the Middle Calcasieu River AOC is predicted to be less than 2.50 ng/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 100% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

TCDD-TEQs in Reference Areas

Integration of the TCDD-TEQs effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to TCDD-TEQs in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental TCDD-TEQ contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to TCDD-TEQ in the Calcasieu reference areas is predicted to be less than 0.428 ng/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 4% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the TCDD-TEQs effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to TCDD-TEQs in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental TCDD-TEQ contamination. The dose predicted to induce 1% effect is 14.9 ng/kg bw/d, while exposure to TCDD-TEQ in

the Calcasieu reference areas is predicted to be less than 0.797 ng/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 75% probability of total daily TCDD-TEQs intake exceeding the Appendix G benchmark of 0.382 ng/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Selenium in Bayou d'Inde AOC

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to selenium in the Bayou d'Inde AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Bayou d'Inde AOC is predicted to be less than 0.0588 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 14% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to selenium in the Bayou

d'Inde AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Bayou d'Inde AOC is predicted to be less than 0.109 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 84% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Selenium in Middle Calcasieu River AOC

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to selenium in the Middle Calcasieu River AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Middle Calcasieu River AOC is predicted to be less than 0.0551 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 13% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to selenium in the Middle Calcasieu River AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Middle Calcasieu River AOC is predicted to be less than 0.104 mg/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 81% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Selenium in Upper Calcasieu River AOC

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to selenium in the Upper Calcasieu River AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Upper Calcasieu River AOC is predicted to be less than 0.0629 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 17% probability of exceeding the Appendix G benchmark, respectively. The

probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to selenium in the Upper Calcasieu River AOC indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Upper Calcasieu River AOC is predicted to be less than 0.118 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 2% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 90% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Selenium in Reference Areas

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to selenium in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Calcasieu reference areas is predicted to be less than 0.0426 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 19% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the selenium effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to selenium in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for mortality due to environmental selenium contamination. The dose predicted to induce 1% effect is 3.30 mg/kg bw/d, while exposure to selenium in the Calcasieu reference areas is predicted to be less than 0.0800 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 0% probability of total daily selenium intake exceeding the Appendix G benchmark of 0.117 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 88% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Total PCBs in Bayou d'Inde AOC

Integration of the total PCBs effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to total PCBs in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental total PCBs contamination. The dose predicted to induce 1% effect is 0.566 mg/kg bw/d, while exposure to total PCBs in the Bayou d'Inde

AOC is predicted to be less than 0.0109 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 100% probability of total daily total PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/d. The lower and upper probability bounds of exposure have a 50% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the total PCBs effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to total PCBs in the Bayou d'Inde AOC indicate that this receptor is at low risk for reproductive effects due to environmental total PCBs contamination. The dose predicted to induce 1% effect is 0.566 mg/kg bw/d, while exposure to total PCBs in the Bayou d'Inde AOC is predicted to be less than 0.0206 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 100% probability of total daily total PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/d. The lower and upper probability bounds of exposure have a 97% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Total PCBs in Reference Areas

Integration of the total PCBs effects curve with the Monte Carlo and probability bounds curves for exposure of the average-sized omnivorous mammals to total PCBs

in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental total PCBs contamination. The dose predicted to induce 1% effect is 0.566 mg/kg bw/d, while exposure to total PCBs in the reference areas is predicted to be less than 0.00189 mg/kg bw/d 98% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that average-sized omnivorous mammals have a 0% probability of total daily total PCBs intake exceeding the Appendix G benchmark of 0.0116 mg/kg bw/d. The lower and upper probability bounds of exposure have a 0% and 61% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Integration of the total PCBs effects curve with the Monte Carlo and probability bounds curves for exposure of the small omnivorous mammals to total PCBs in the Calcasieu Estuary reference areas indicate that this receptor is at low risk for reproductive effects due to environmental total PCBs contamination. The dose predicted to induce 1% effect is 0.566 mg/kg bw/d, while exposure to total PCBs in the reference areas is predicted to be less than 0.00369 mg/kg bw/d 99% of the time according to the Monte Carlo analysis.

The Monte Carlo analysis indicated that small omnivorous mammals have a 15% probability of total daily total PCBs intake exceeding the Appendix G benchmark of 0.00272 mg/kg bw/d. The lower and upper probability bounds of exposure have a 4% and 100% probability of exceeding the Appendix G benchmark, respectively. The probabilities of COCs exceeding the Appendix G benchmarks are summarized in Table I1-4.

Historical data

Levels of Aroclor 1254 in tissues of fish collected from CH2M Hill's Calcasieu Estuary Biological Monitoring Program were consistent with levels found in the Phase II Sampling Program. Levels in whole body determined in 2001 during Phase II Sampling and levels in fillet recorded since 1991 by CH2M Hill were used for statistical analysis. For comparison, fillet concentrations were estimated for the samples collected from the Phase II Sampling Program using the following equation:

$$C_f = C_{wb} / 2.3 \qquad (3)$$

where, C_{wb} is whole-body concentration and C_f is fillet concentration (SAIC 1993).

Annual geometric mean concentrations in fillet of red drum, black drum, spotted seatrout, sand seatrout and southern flounder were calculated for the four AOCs.

The geometric mean concentration of Aroclor 1254 in fillet collected from the Bayou d'Inde AOC during the Phase II Sampling Program was 0.016 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.230 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.028 mg/kg to 0.133 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 1.080 mg/kg, respectively (Figure I1-30).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Middle Calcasieu River AOC during the Phase II Sampling Program was 0.013 mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.317 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.008 mg/kg to 0.031 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.221 mg/kg, respectively (Figure I1-31).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Upper Calcasieu River AOC during the Phase II Sampling Program was 0.013 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.478 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.040 mg/kg and the minimum and maximum concentrations were 0.005 mg/kg and 0.232 mg/kg, respectively (Figure I1-32).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Calcasieu Estuary reference areas during the Phase II Sampling Program was 0.006 mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.029 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.016 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.378 mg/kg, respectively (Figure I1-33).

The comparison of historical data sets between the Phase II Sampling Program and CH2M Hill's Biological Monitoring Program showed that there was less than one order of magnitude difference in levels of total PCBs in fish tissue between the ten years of historical data and data collected in the Phase II Sampling Program. In most cases, the difference was less than four fold. This demonstrates that the results of the ecological risk assessment for carnivorous fish using data from the Phase II Sampling Program are likely to be temporally representative.

4.0 Uncertainty Analysis

There are a number of sources of uncertainty in the assessments of risk to omnivorous mammals, including uncertainties in the conceptual model, and in the exposure, effects, and risk assessments. As each of these sources of uncertainty can influence the estimates of risk, it is important to describe and, when possible, quantify the magnitude and direction of such uncertainties. In this way, it is possible to evaluate the level of confidence that can be placed in the assessments conducted. The uncertainties associated with the assessment of risks to omnivorous mammals are described in the following sections.

Uncertainties Associated with the Conceptual Model - The conceptual model is intended to define the linkages between stressors, potential exposure, and predicted effects on ecological receptors. As such, the conceptual model provides the scientific basis for selecting assessment and measurement endpoints to support the risk assessment process. Potential uncertainties arise from lack of knowledge regarding ecosystem functions, failure to adequately address spatial and temporal variability in the evaluations of sources, fate, and effects, omission of stressors, and overlooking secondary effects (USEPA 1998). The types of uncertainties associated with the conceptual model that links contaminant sources to effects on omnivorous mammals include those associated with the identification of COCs, environmental fate and transport of COCs, exposure pathways, receptors at risk, and ecological effects. Of these, the identification of exposure pathways probably represents the primary source of uncertainty in the conceptual model. In this assessment, it was assumed that exposure to contaminated food represented the most important pathway for exposing omnivorous mammals to COCs. Although unlikely to be important, other pathways could contribute to exposure and perhaps increase risk somewhat.

Uncertainties Associated with the Exposure Assessment - The exposure assessment is intended to describe the actual or potential co-occurrence of stressors with receptors. As such, the exposure assessment identifies the exposure pathways and the intensity and extent of contact with stressors for each receptor or group of receptors at risk. There are a number of potential sources of uncertainty in the exposure assessment, including measurement errors, extrapolation errors, and data gaps.

In this assessment, chemical analyses of tissue residues in fish and invertebrates were used to evaluate exposure of omnivorous mammals to COCs. Analytical errors and descriptive errors represent potential sources of uncertainty in this measurement.

Three approaches were used to address concerns relative to these sources of uncertainty. First, analytical errors were evaluated using information on the accuracy, precision, and detection limits (DL) generated to support the Phase I and Phase II sampling programs. The results of this analysis indicated that most of the data used in this assessment met the project data quality objectives (see Appendix B1 for more details). Second, all data entry, data translation, and data manipulations were audited to ensure their accuracy. Data auditing involved 10% number-for-number checks against the primary data source initially, increasing to 100% number-for-number checks if significant errors were detected in the initial auditing step. Finally, statistical analyses of data were conducted to evaluate data distributions, identify appropriate summary statistics, and evaluate variability in the observations. Using these techniques, we were able to identify outliers and, if the outliers were due to an error, correct the outlier values.

According to the Monte Carlo sensitivity analyses, the FMR slope and power term were among the most influential variables driving the predicted intake rates. Unfortunately, a precise estimate of the FMR was not possible as suitable measured

metabolic rates for omnivorous mammals were not available in the literature. Instead, the FMR for omnivorous mammals was estimated using allometric equations. This introduced some degree of uncertainty into the exposure estimates because the allometric relationships were not only associated with some fitting error, but also were based on many mammal species, some of which were very different from those represented here. However, given the lack of empirical data on species specific to the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric model rather than the empirical data.

Other sensitive variables that influenced the exposure estimates included the gross energy of food and the food assimilation efficiency. These variables also were somewhat uncertain because no feeding studies were specifically performed in the Calcasieu Estuary on the species of interest. Rather, diet compositions were matched to those reported in the literature from other geographical locations. As a consequence, the quantification of food gross energy and assimilation efficiency was limited to the fish food group, without considering specific fish species. Furthermore, the estimates were uncertain, because they were approximated using gross energy and assimilation efficiency data for generic fish. Prey tissue sample sizes were small for many of the COC analyses in the AOCs, thus adding to the uncertainty in the omnivorous mammal exposure characterization.

Uncertainties in the Effects Assessment - The effects assessment is intended to describe the effects caused by stressors, link them to the assessment endpoints, and evaluate how effects change with fluctuations in the levels (i.e., concentrations or doses) of the various stressors. There are several sources of uncertainty in the assessment of effects including measurement errors, extrapolation errors, model fit errors, and data gaps.

The greatest source of uncertainty for the effects characterization is the lack of toxicity studies in which the representative species were dosed with methylmercury, PCBs, TEQs, and selenium. There were no toxicity studies available that treated raccoons, or another suitable wild omnivorous mammal, with doses of these COCs. Studies involving surrogate species, namely rats and mice, were used instead. This added another degree of uncertainty because it is not known whether laboratory raised and tested animals have the same sensitivity as those living in the wild. Studies of the reproductive success of omnivorous mammals performed in situ in the Calcasieu Estuary were also not available. Such site specific field studies would have been more able to account for the specific characteristics of the Calcasieu Estuary ecosystem.

Another significant source of uncertainty in the risk assessment for omnivorous mammals is the lack of information available on abundance of omnivorous mammals across chemical gradients in the Calcasieu Estuary, and lack of toxicity studies on the responses of omnivorous mammals fed prey collected from the estuary.

5.0 Conclusions

The risk characterization results indicate that there is little chance that exposure to any of the relevant COCs will cause significant adverse effects to omnivorous mammals foraging in the Calcasieu Estuary. This statement holds true when account is taken of the smallest animals in the guild, and even when sources of incertitude are considered. There are, however, several limitations of the probabilistic risk analyses that influence our confidence regarding the above risk statements. These include:

- The sensitivity analyses for the Monte Carlo simulations indicated that the most important input variable was the power and slope terms for the free metabolic rate (*FMR*) equation. The *FMR* used in the analyses was based on the allometric equation from Nagy (1987). No *in situ* measurements of this variable are available for omnivorous mammals. The potential magnitude and direction of the uncertainty associated with lack of empirical data on metabolic rate are unknown. We did, however, investigate the possible consequences of the uncertainty in this variable due to model error (i.e., the error associated with the lack of fit of the allometric model that relates *FMR* to body weight) in the probability bounds analysis. This source of uncertainty did not strongly impact our conclusions regarding risk;

- Sample size for COCs in fish and invertebrate tissues was generally limited. Although we accounted for this source of uncertainty in our analyses, it is possible that additional data would substantially change the distribution for this variable;

- Omnivorous mammals feed on aquatic plants and terrestrial prey as well. Data on levels of COCs in these food items, however, were not available. It seems likely that daily COC exposure would change for omnivorous mammals were this taken into account. The amount of the potential increase is unknowable; and,

- The effects analyses pointed out several key sources of uncertainty. First, no data were available for omnivorous mammalian species found in the Calcasieu Estuary. Second, differing environmental conditions

between the laboratory and the field introduces uncertainty to the estimation of effects doses.

The above described limitations are common to wildlife risk assessments and indicate the value of having other lines of evidence to help characterize risks. Biological surveys and ambient toxicity testing are two such lines of evidence. No *in situ* or whole media feeding studies are available, however, for omnivorous mammals in the Calcasieu Estuary. Formal biological surveys that relate degree of COC contamination to abundances of different omnivorous mammalian species have not been conducted. While the evidence presented certainly cannot be used to rule out the possibility that COCs are causing adverse effects to omnivorous mammals in the Calcasieu Estuary, it does seem unlikely that COCs are causing widespread impacts.

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Tables

Table I1-1. Results of the deterministic risk assessment for omnivorous mammals in the Calcasieu Estuary.

Contaminant of Concern (COC)	Area	Risk Quotient	Proceed to Probabilistic Assessment
Mercury	Bayou d'Inde	1.5	Yes
	Middle Calcasieu River	0.3	No
	Upper Calcasieu River	0.3	No
	Reference	0.1	Yes
Toxic equivalents (TEQs)	Bayou d'Inde	17.6	Yes
	Middle Calcasieu River	14.7	Yes
	Upper Calcasieu River	4.97	No
	Reference	6.29	Yes
Selenium	Bayou d'Inde	1.6	Yes
	Middle Calcasieu River	1.2	Yes
	Upper Calcasieu River	1.1	Yes
	Reference	0.8	Yes
Total polychlorinated biphenyls (PCBs)	Bayou d'Inde	45.9	Yes
	Middle Calcasieu River	1.7	No
	Upper Calcasieu River	6.3	No
	Reference	25.8	Yes

Table I1-2. Monte Carlo input variables.

Variable			Distribution	Parameters
Body weight: average-sized species (BW; g)			normal	mean = 2602; SD = 338
Body weight: small species (BW; g)			normal	mean = 55.0; SD = 7.15
Free Metabolic Rate (FMR; Kcal/kg bw/day)			$FMR=aBW^b$	
a = FMR slope term			normal	log mean = 0.525; log SD = 0.057
b = FMR power term			normal	mean = 0.813; SD = 0.023
Assimilation Efficiency - Fish (AE_f, unitless)			beta	á = 60.0; â = 7.00; scale = 1.00
Assimilation Efficiency - Invertebrates (AE_i, unitless)			beta	á = 60.0; â = 7.00; scale = 1.00
Gross Energy - Fish (GE_f; Kcal/kg)			lognormal	mean = 1200; SD = 240
Gross Energy - Invertebrates (GE_i; Kcal/kg)			lognormal	mean = 967; SD = 193
Proportion of diet - Fish (%)			point estimate	16.7
Proportion of diet - Invertebrates (%)			point estimate	16.7
Contaminants of Concern (COCs)				
COCs	**Area**	**Tissue Classification**		
Mercury	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.0373; SD = 0.000434
		C_{fish} (mg/kg ww)	lognormal	mean = 0.189; SD = 0.00638
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.00751; SD = 0.0000587
		C_{fish} (mg/kg ww)	lognormal	mean = 0.0244; SD = 0.000306
Tetrachlorodibenzo-*p* -dioxin (TCDD) and equivalents	Bayou d'Inde	C_{invert} (ng/kg ww)	lognormal	mean = 22.2; SD = 0.556
		C_{fish} (ng/kg ww)	lognormal	mean = 29.6; SD = 0.459
	Middle Calcasieu River	C_{invert} (ng/kg ww)	lognormal	mean = 7.29; SD = 0.0230
		C_{fish} (ng/kg ww)	lognormal	mean = 16.2; SD = 0.434

Table I1-2. Monte Carlo input variables.

Variable			Distribution	Parameters
COCs (cont.)	**Area (cont.)**	**Tissue Classification (cont.)**		
TCDD and equivalents (cont.)	Reference Areas	C_{invert} (ng/kg ww)	point estimate	0
		C_{fish} (ng/kg ww)	lognormal	mean = 7.64; SD = 0.119
Selenium	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.460; SD = 0.00140
		C_{fish} (mg/kg ww)	lognormal	mean = 0.562; SD = 0.00620
	Middle Calcasieu River	C_{invert} (mg/kg ww)	lognormal	mean = 0.463; SD = 0.00551
		C_{fish} (mg/kg ww)	lognormal	mean = 0.499; SD = 0.0133
	Upper Calcasieu River	C_{invert} (mg/kg ww)	lognormal	mean = 0.575; SD = 0.00505
		C_{fish} (mg/kg ww)	lognormal	mean = 0.509; SD = 0.00625
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.423; SD = 0.00594
		C_{fish} (mg/kg ww)	lognormal	mean = 0.304; SD = 0.00571
Total polychlorinated biphenyls (PCBs)	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.0328; SD = 0.000732
		C_{fish} (mg/kg ww)	lognormal	mean = 0.162; SD = 0.0148
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.0100; SD = 0.000268
		C_{fish} (mg/kg ww)	lognormal	mean = 0.0230; SD = 0.00258

Table I1-3. Probability bounds input variables.

Variable			Distribution	Parameters
Body weight: average-sized species (BW; g)			normal	mean = 2602; SD = 338
Body weight: small species (BW; g)			normal	mean = 55.0; SD = 7.15
Free Metabolic Rate (FMR; Kcal/kg bw/day)			$FMR=aBW^b$	
a = FMR slope term			normal	log mean = 0.525; log SD = 0.057
b = FMR power term			normal	mean = 0.813; SD = 0.023
Assimilation Efficiency - Fish (AE_f, unitless)			minmaxmean	0.75, 0.98, 0.90
Assimilation Efficiency - Invertebrates (AE_i, unitless)			minmaxmean	0.75, 0.98, 0.90
Gross Energy - Fish (GE_f; Kcal/kg)			lognormal	mean = 1200; SD = 240
Gross Energy - Invertebrates (GE_i; Kcal/kg)			lognormal	mean = 967; SD = 193
Proportion of diet - Fish (%)			point estimate	16.7
Proportion of diet - Invertebrates (%)			point estimate	16.7
Contaminants of Concern (COCs)				
COCs	**Area**	**Tissue Classification**		
Mercury	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.0438; SD = 0.00695
		C_{fish} (mg/kg ww)	lognormal	mean = 0.196; SD = 0.0217
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.00998; SD = 0.00238
		C_{fish} (mg/kg ww)	lognormal	mean = 0.0312; SD = 0.00724
Tetrachlorodibenzo-*p*-dioxin (TCDD) and equivalents	Bayou d'Inde	C_{invert} (ng/kg ww)	uniform	min = 11.9; max = 43.0
		C_{fish} (ng/kg ww)	lognormal	mean = 34.0; SD = 5.86

Table I1-3. Probability bounds input variables.

Variable			Distribution	Parameters
COCs (cont.)	**Area (cont.)**	**Tissue Classification (cont.)**		
TCDD and equivalents (cont.)	Middle Calcasieu River	C_{invert} (ng/kg ww)	uniform	min = 6.28; max = 7.90
		C_{fish} (ng/kg ww)	uniform	min = 2.92; max = 23.1
	Reference Areas	C_{invert} (ng/kg ww)	point estimate	0
		C_{fish} (ng/kg ww)	uniform	min = 3.99; max = 9.88
Selenium	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.550; SD = 0.0688
		C_{fish} (mg/kg ww)	lognormal	mean = 0.600; SD = 0.0427
	Middle Calcasieu River	C_{invert} (mg/kg ww)	lognormal	mean = 0.548; SD = 0.0965
		C_{fish} (mg/kg ww)	lognormal	mean = 0.560; SD = 0.117
	Upper Calcasieu River	C_{invert} (mg/kg ww)	lognormal	mean = 0.653; SD = 0.0779
		C_{fish} (mg/kg ww)	lognormal	mean = 0.558; SD = 0.0582
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.567; SD = 0.168
		C_{fish} (mg/kg ww)	lognormal	mean = 0.636; SD = 0.0816
Total polychlorinated biphenyls (PCBs)	Bayou d'Inde	C_{invert} (mg/kg ww)	lognormal	mean = 0.0367; SD = 0.00784
		C_{fish} (mg/kg ww)	lognormal	mean = 0.132; SD = 0.0209
	Reference Areas	C_{invert} (mg/kg ww)	lognormal	mean = 0.0131; SD = 0.00483
		C_{fish} (mg/kg ww)	lognormal	mean = 0.0303; SD = 0.0187

Table I1-4. Probabilities of exposure of omnivorous mammals to contaminants of concern (COCs) exceeding Appendix G ecological risk assessment benchmarks in the Calcasieu Estuary.

COC and Location	Probability of Exposure Exceeding Benchmark (%)					
	Average-Sized Omnivorous Mammal			Small Omnivorous Mammal		
	LPB	***FOMC***	***UPB***	***LPB***	***FOMC***	***UPB***
Mercury						
Bayou d'Inde	0	3	78	5	80	100
Reference Areas	0	0	1	0	0	4
TCDD and Equivalents						
Bayou d'Inde	24	100	100	100	100	100
Middle Calcasieu River	0	99	100	0	100	100
Reference Areas	0	4	100	0	75	100
Selenium						
Bayou d'Inde	0	0	14	0	0	84
Middle Calcasieu River	0	0	13	0	0	81
Upper Calcasieu River	0	0	17	0	2	90
Reference Areas	0	0	19	0	0	88
PCBs						
Bayou d'Inde	50	100	100	97	100	100
Reference Areas	0	0	61	4	15	100

LPB = Lower Probability Bound; FOMC = First Order Monte Carlo; UPB = Upper Probability Bound;
TCDD = tetrachlorodibenzo-*p* -dioxin; PCBs = polychlorinated biphenyls.

Figures

Figure I1-1. Overview of approach used to assess exposure of omnivorous mammals to contaminants of concern (COCs) in the Calcasieu Estuary.

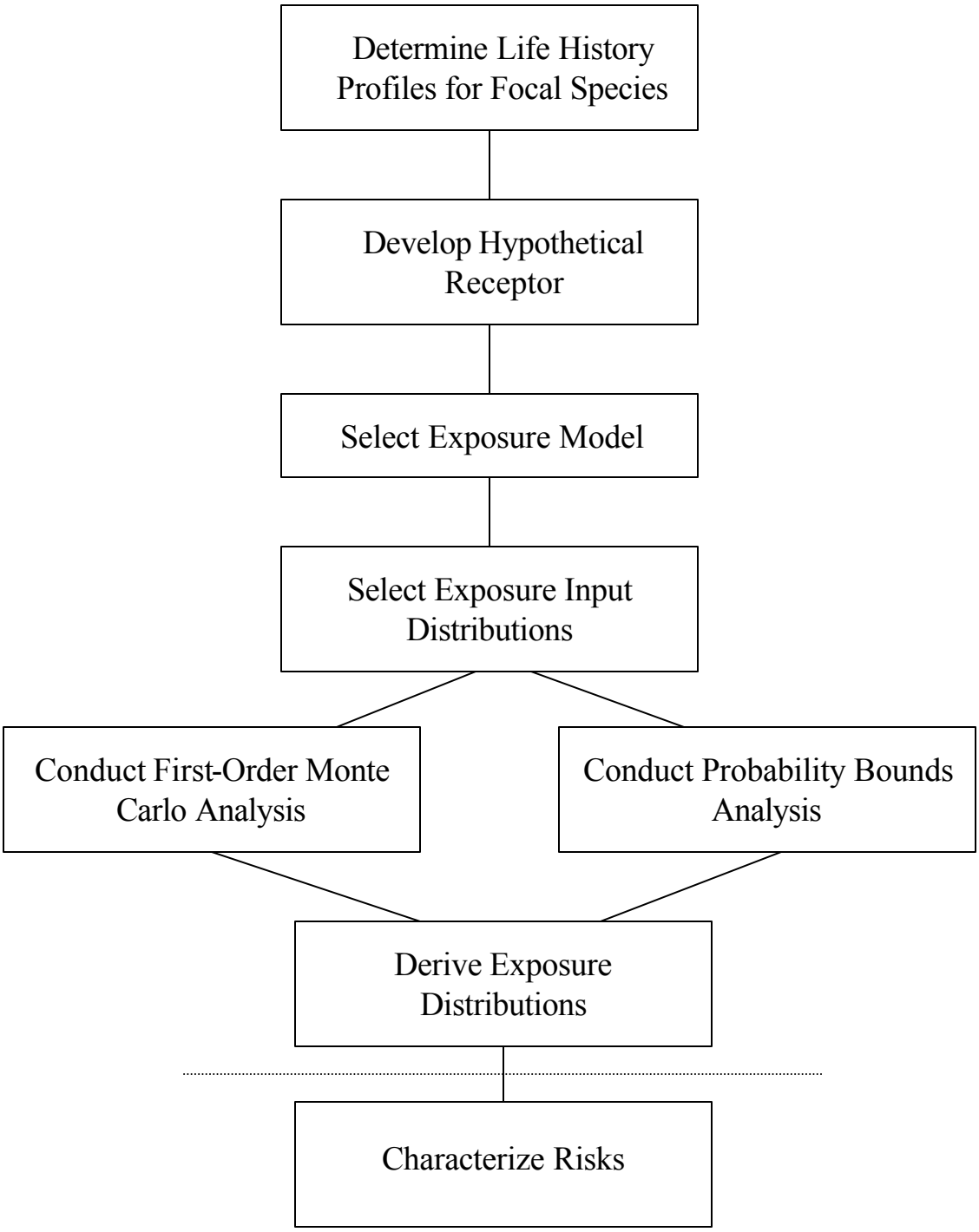

Figure I1-2. Overview of approach used to assess the effects of omnivorous mammals exposed to contaminants of concern (COCs) in the Calcasieu Estuary.

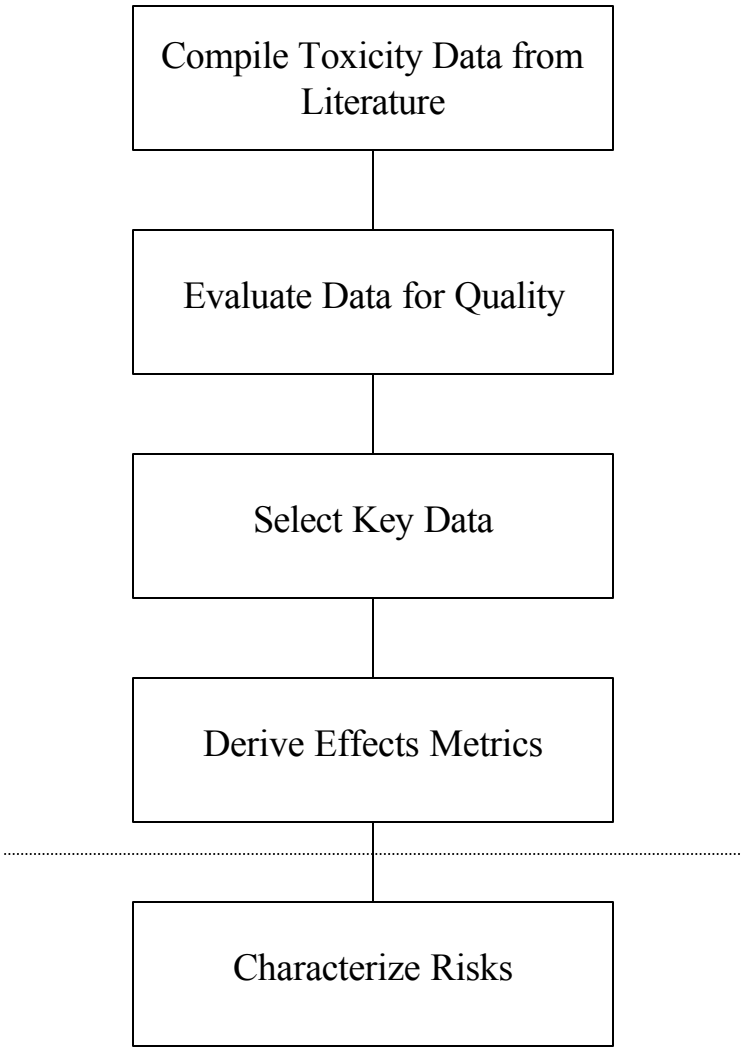

Figure I1-3. Overview of approach used to assess the risks of omnivorous mammals exposed to contaminants of concern (COCs) in the Calcasieu Estuary.

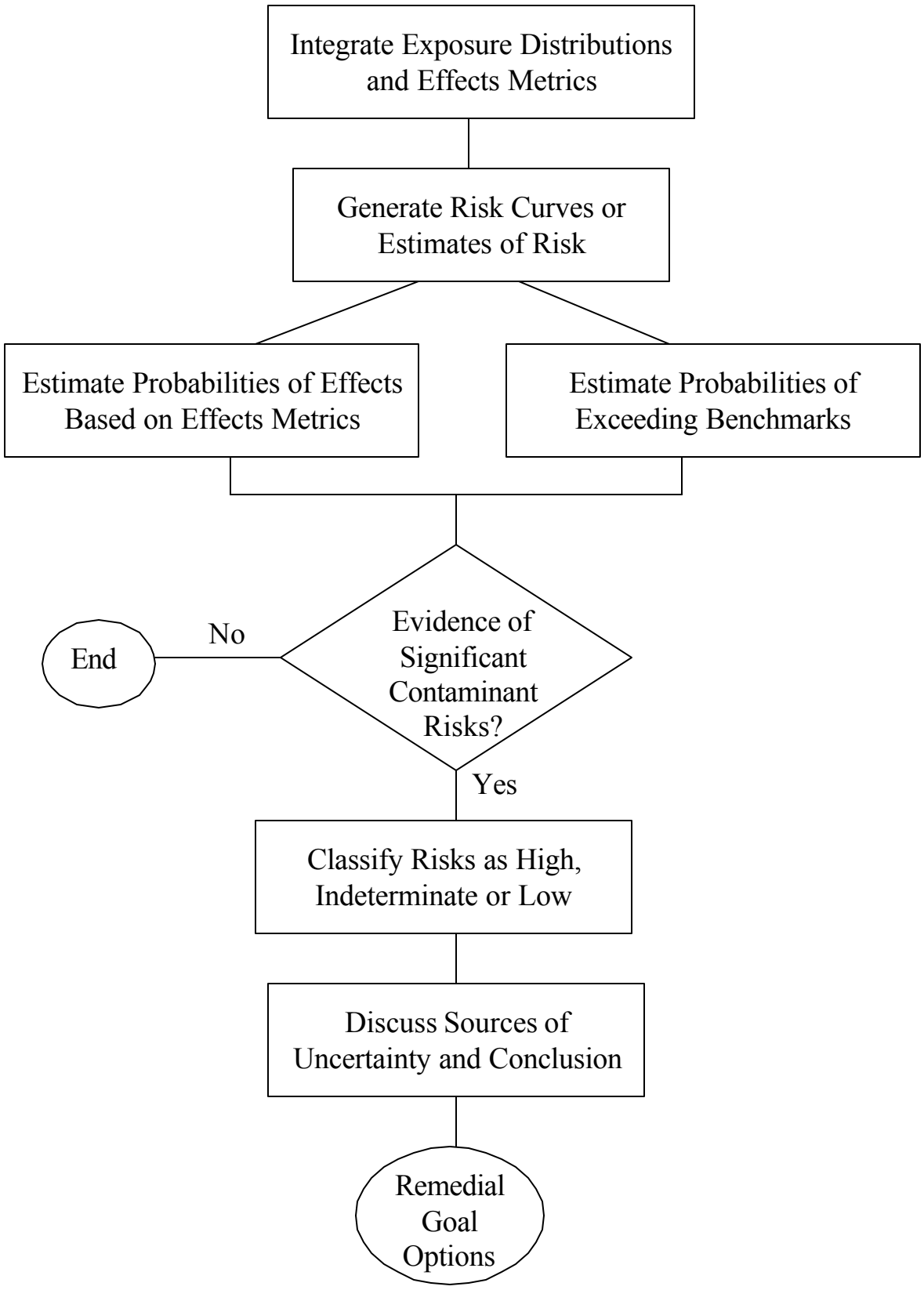

Figure I1-4. Dose-response curve for reproductive fecundity of mice treated orally with methyl mercuric chloride during gestation.

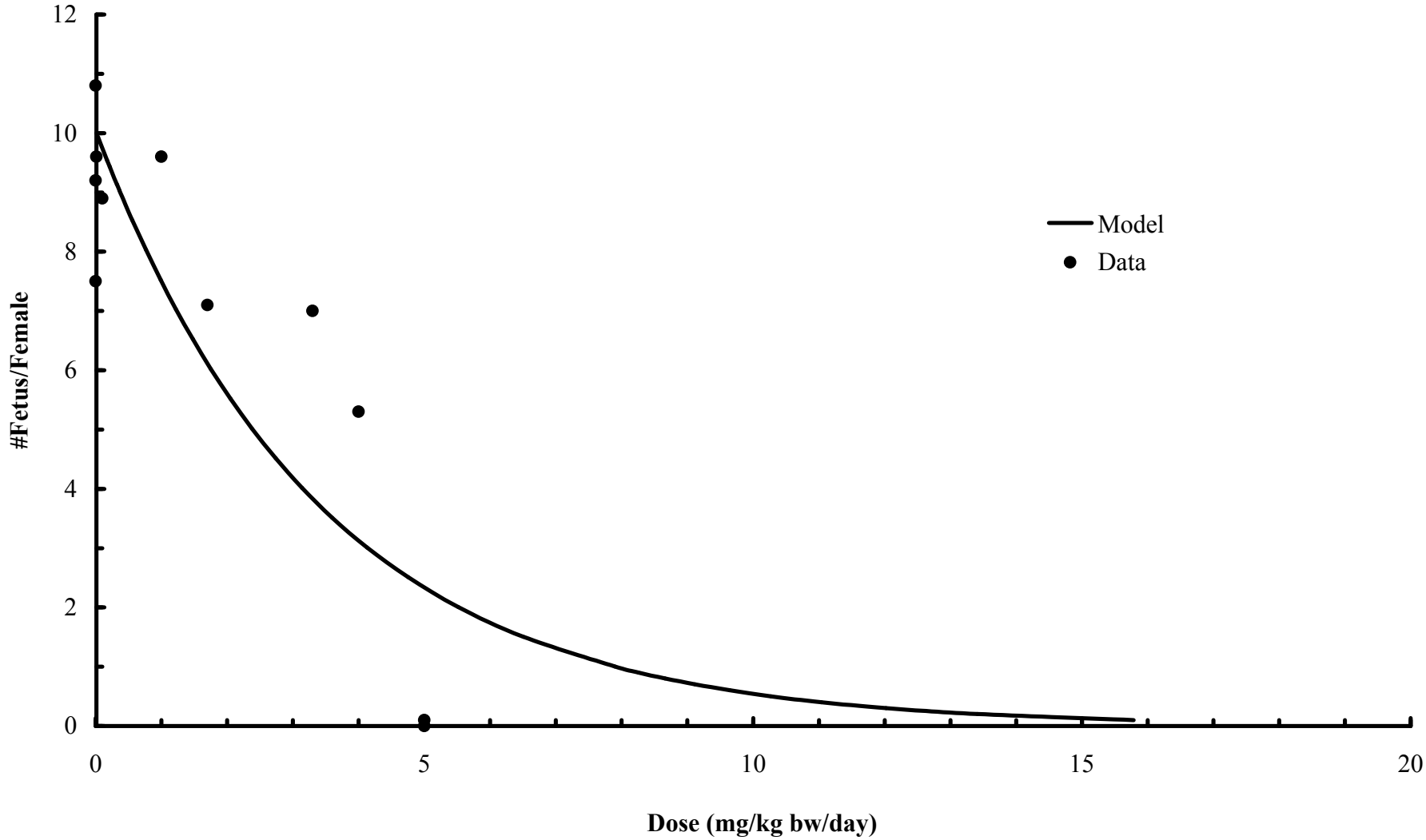

Figure I1-5. Dose-response curve for reproductive fecundity of rats treated orally with TCDD during gestation.

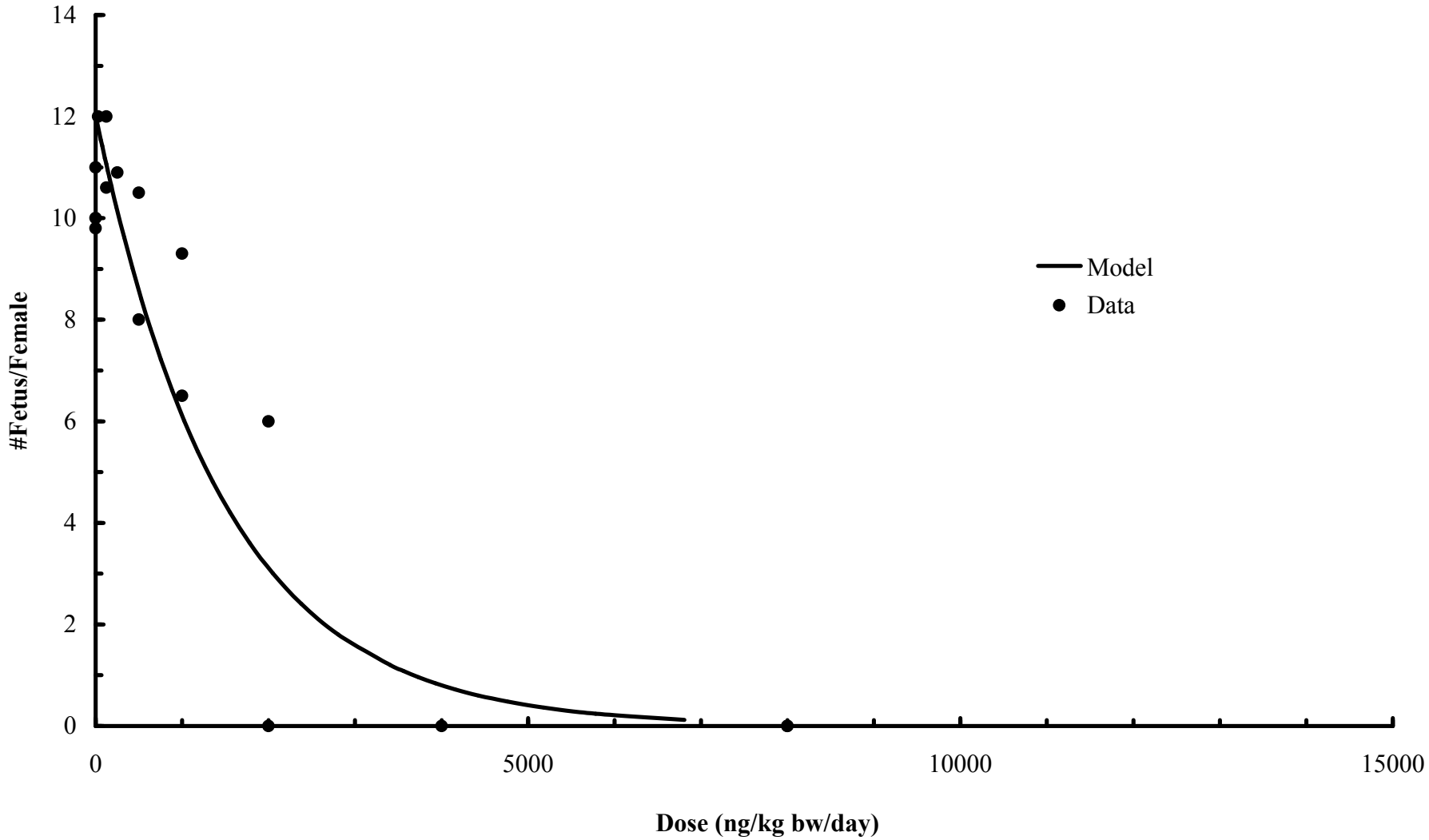

Figure I1-6. Dose-response curve for survival of rats treated orally with sodium selenite.

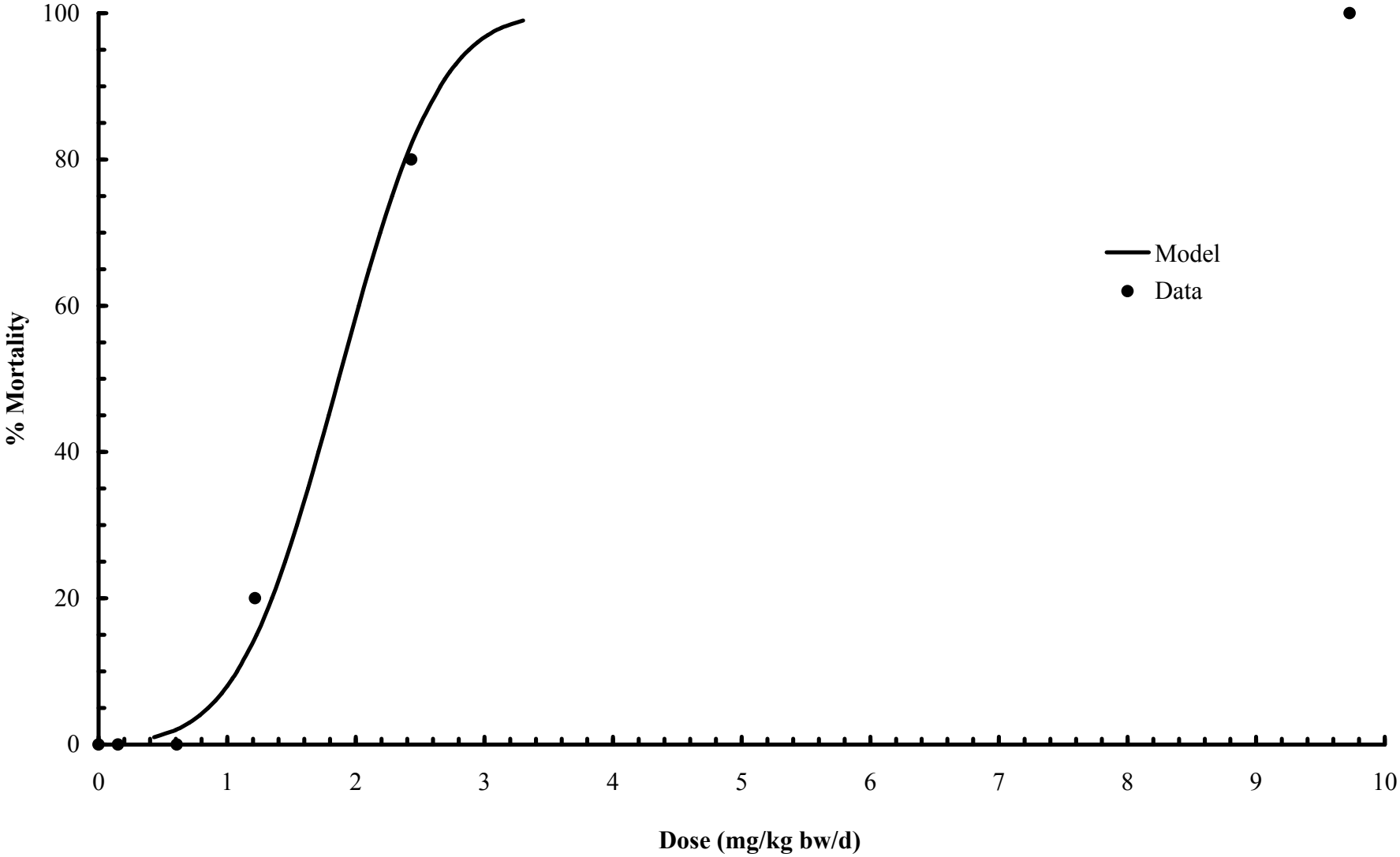

Figure I1-7. Dose-response curve for reproductive fecundity of rats treated orally with Aroclor 1254 during gestation.

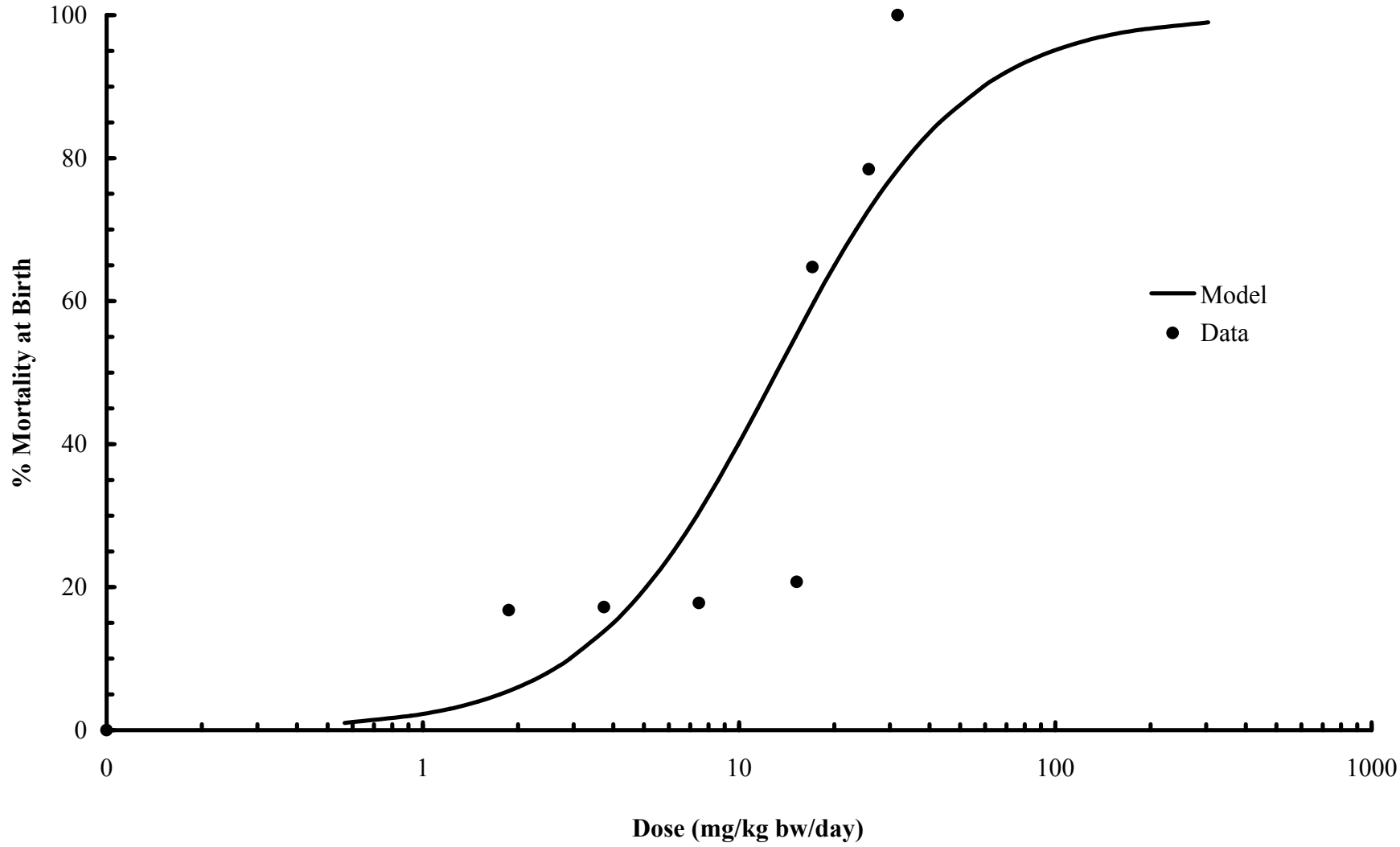

Figure I1-8. Exposure of hypothetical average-sized omnivorous mammals to mercury in Bayou d'Inde AOC.

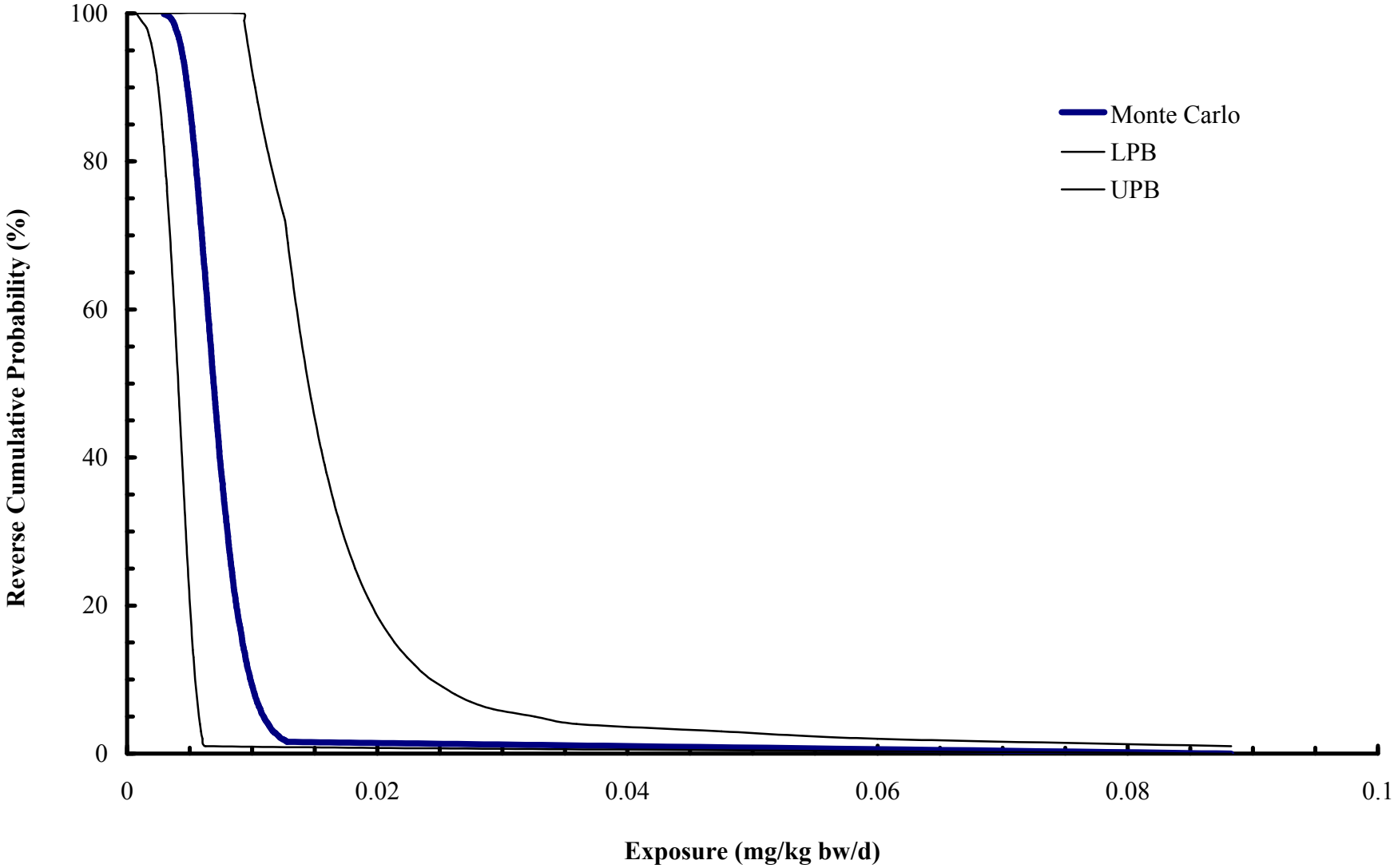

Figure I1-9. Exposure of hypothetical small omnivorous mammals to mercury in Bayou d'Inde AOC.

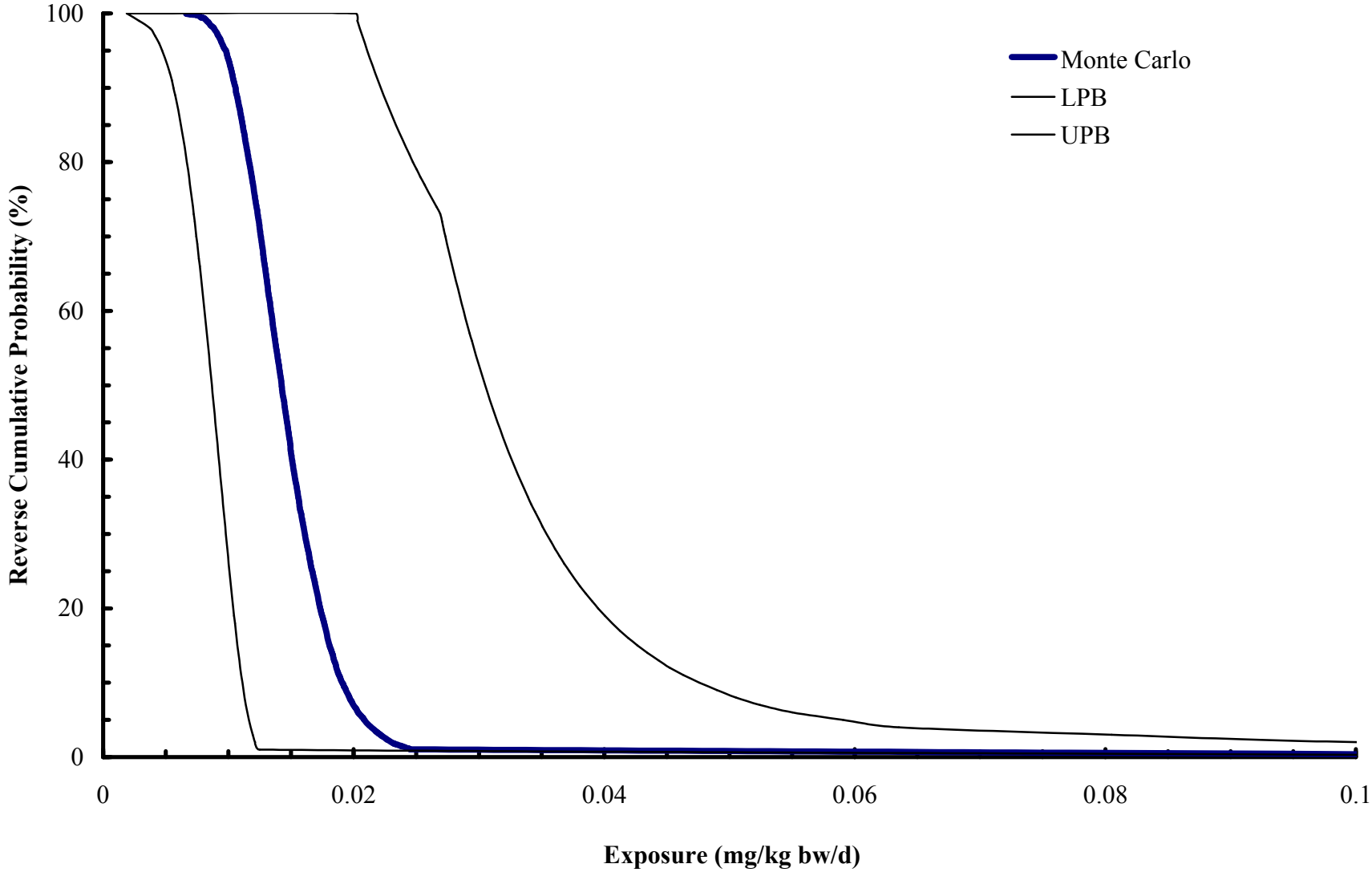

Figure I1-10. Exposure of hypothetical average-sized omnivorous mammals to mercury in the Calcasieu reference areas.

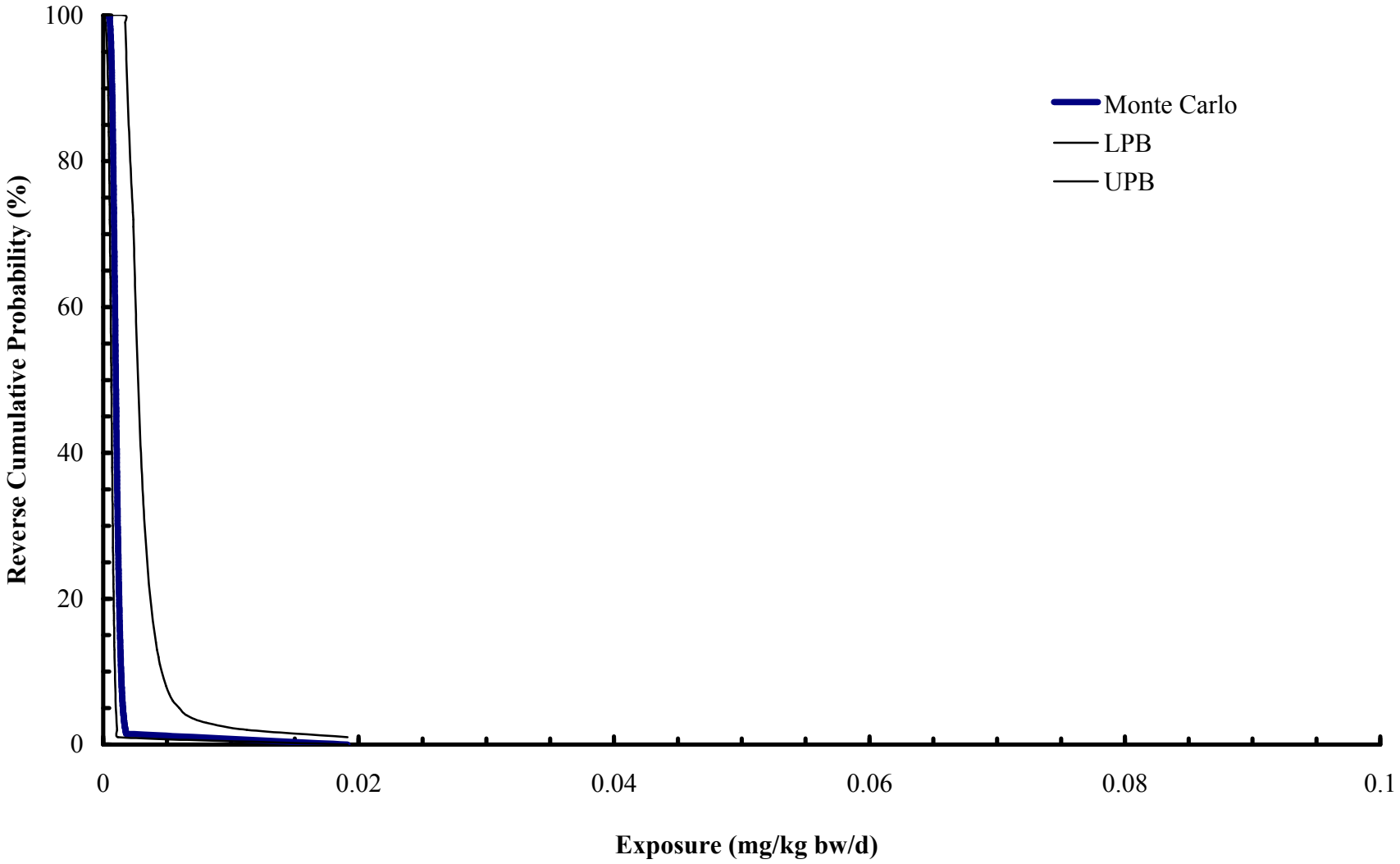

Figure I1-11. Exposure of hypothetical small omnivorous mammals to mercury in the Calcasieu reference areas.

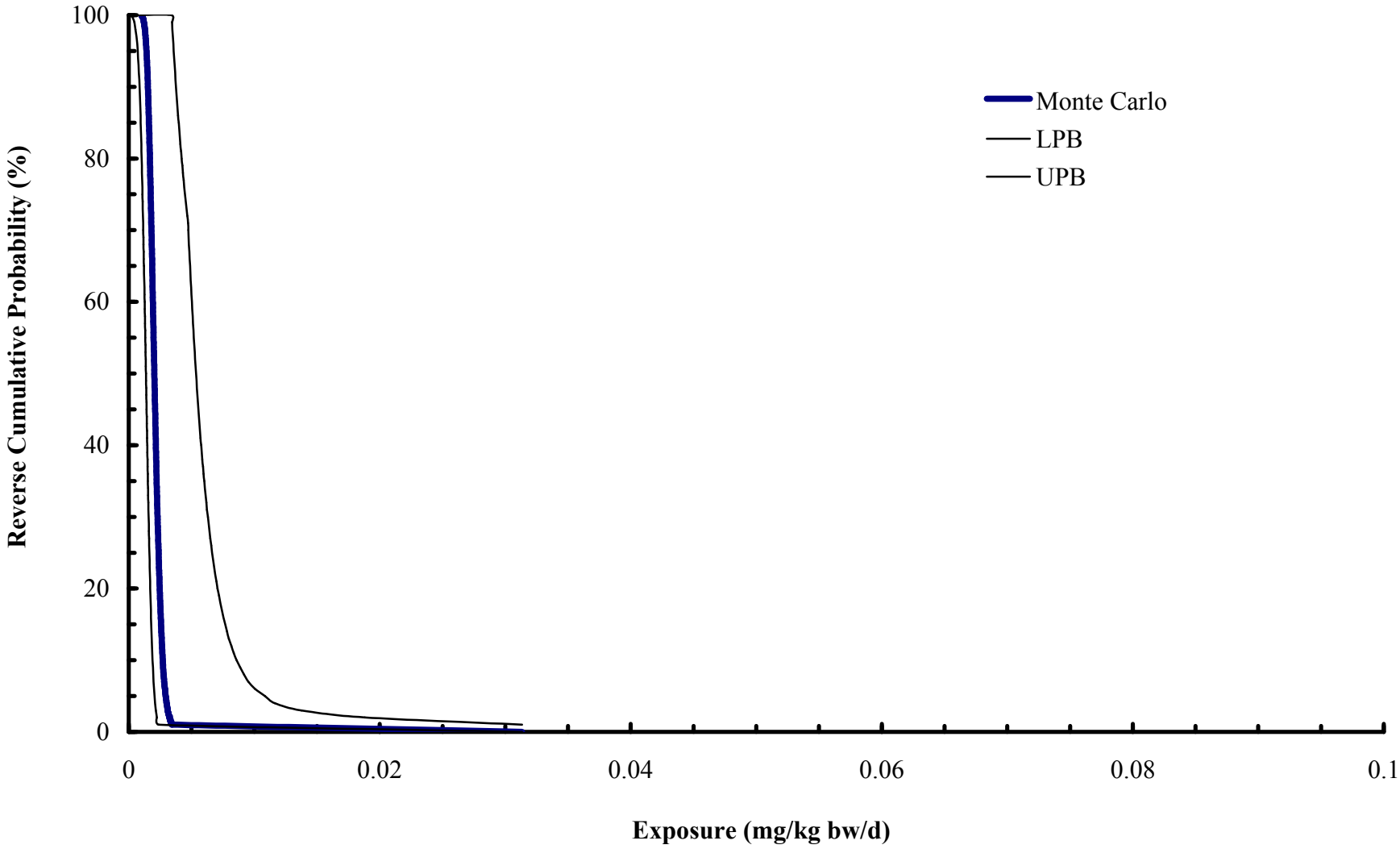

Figure I1-12. Exposure of hypothetical average-sized omnivorous mammals to TCDD and equivalents in Bayou d'Inde AOC.

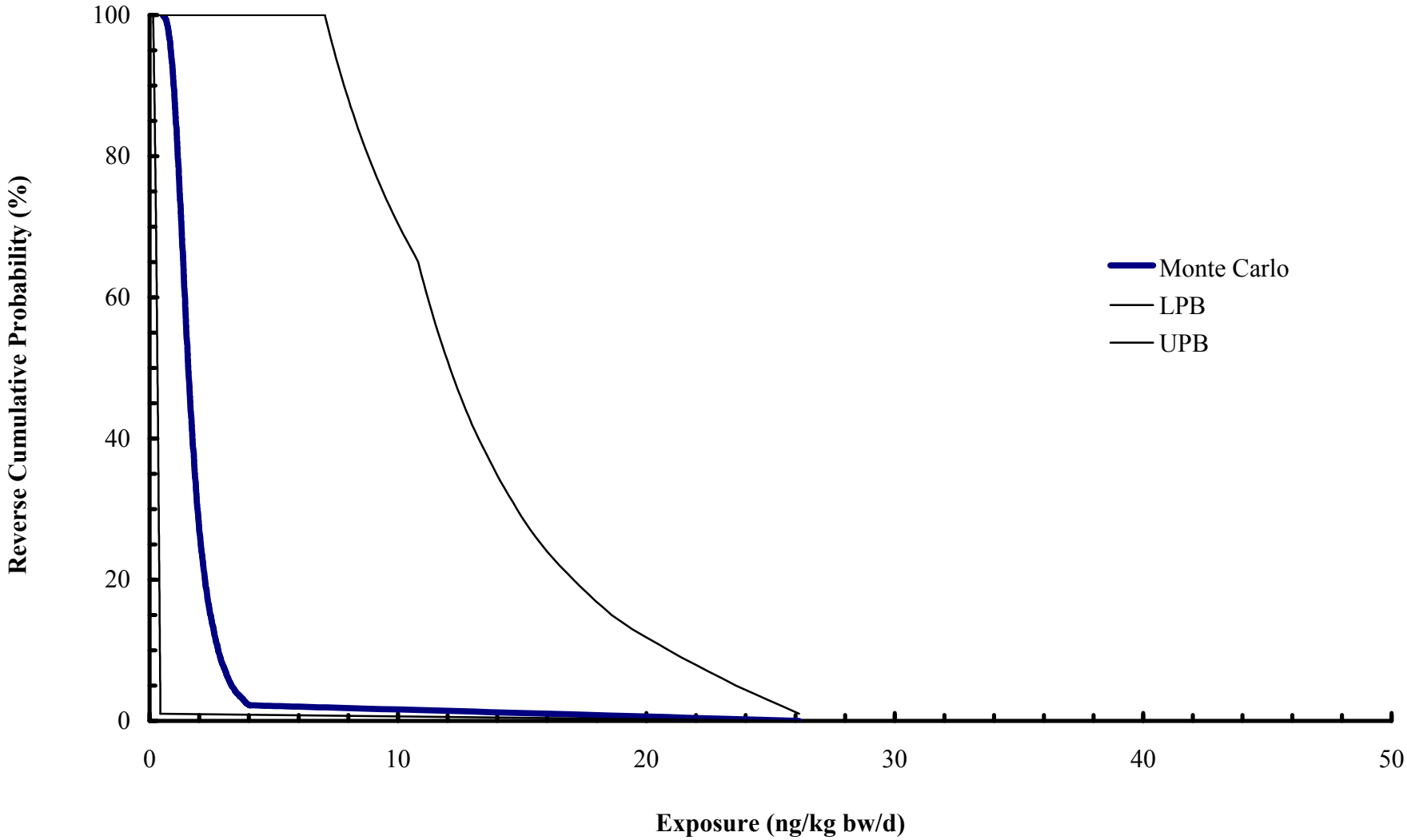

Figure I1-13. Exposure of hypothetical small omnivorous mammals to TCDD and equivalents in Bayou d'Inde AOC.

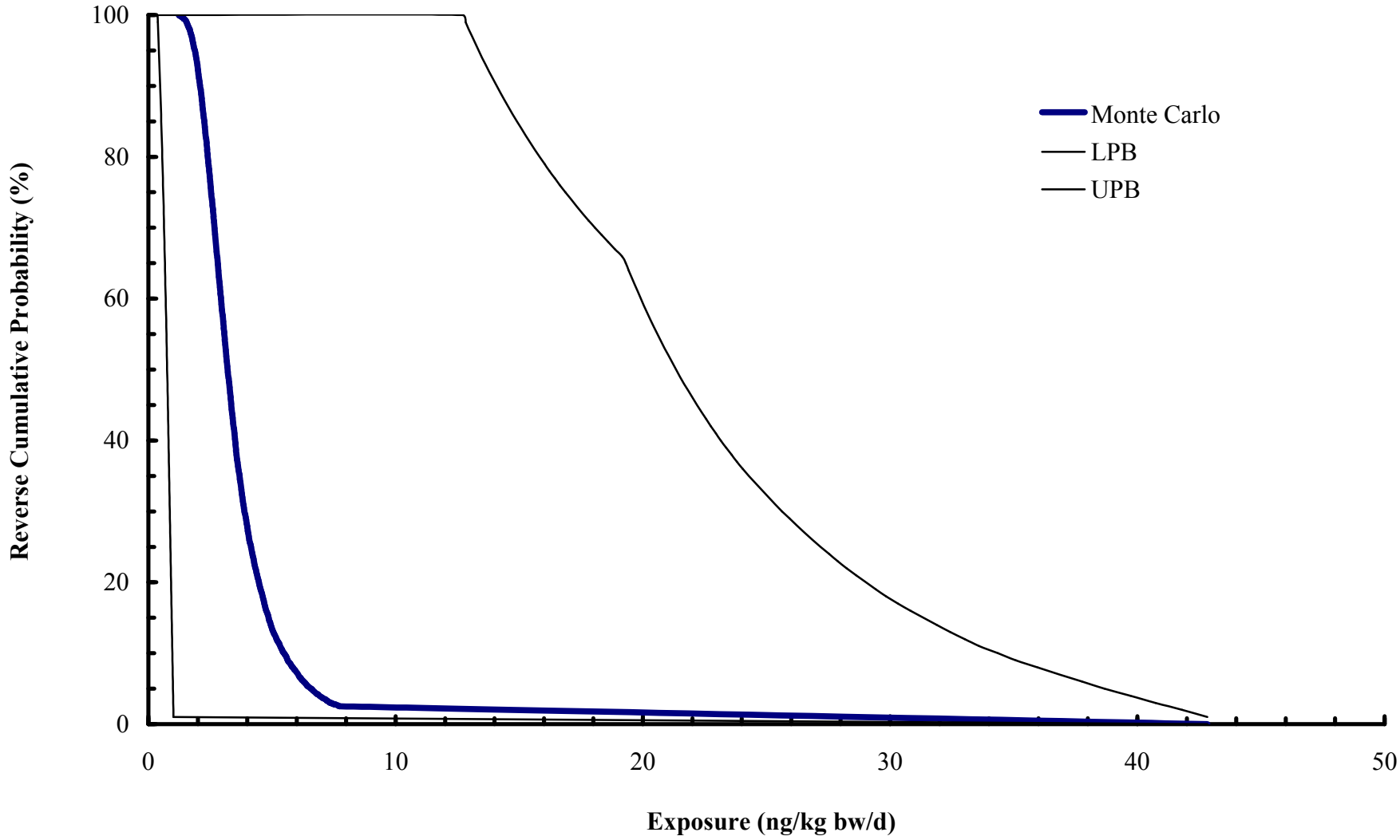

Figure I1-14. Exposure of hypothetical average-sized omnivorous mammals to TCDD and equivalents in Middle Calcasieu River AOC.

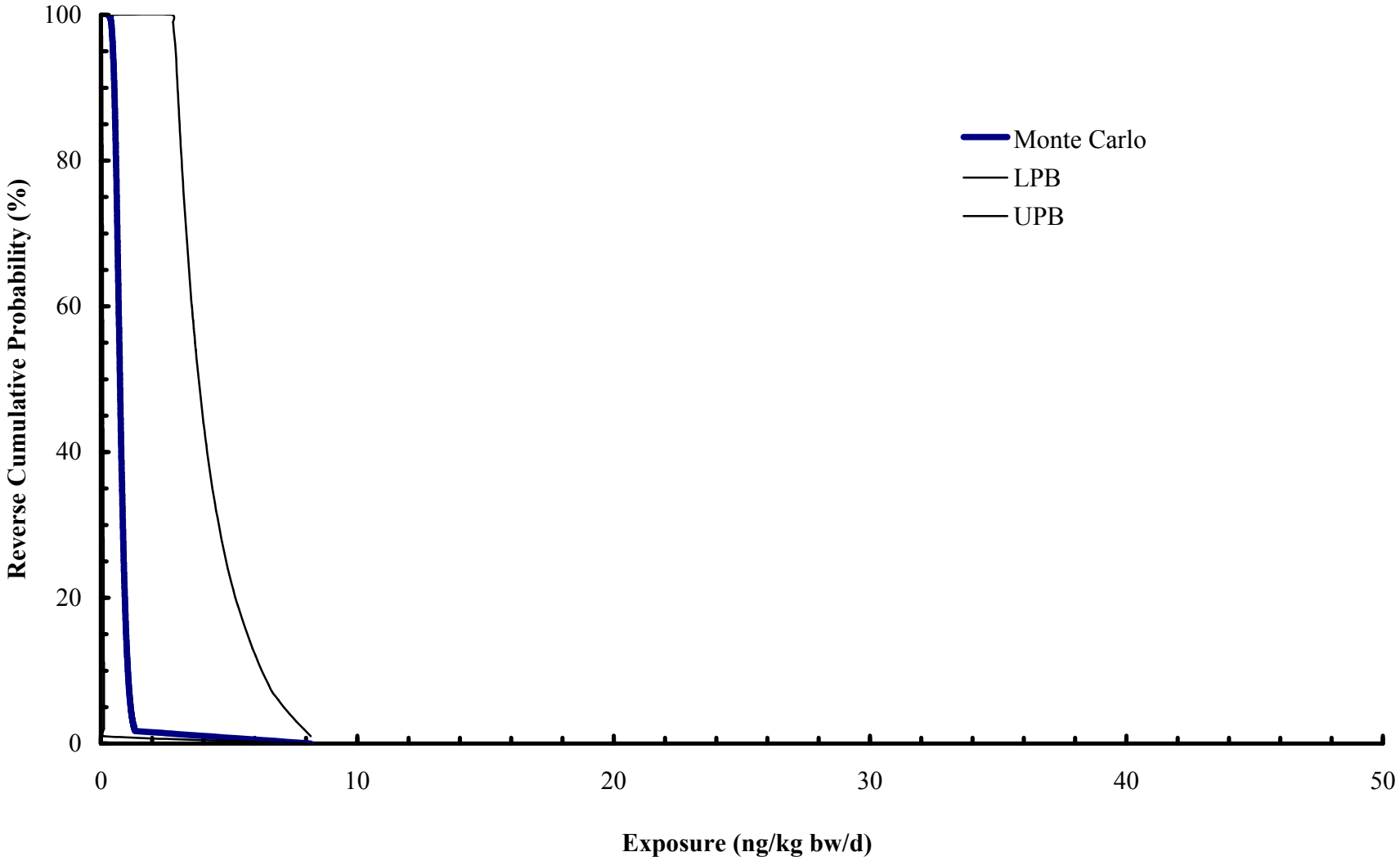

Figure I1-15. Exposure of hypothetical small omnivorous mammals to TCDD and equivalents in Middle Calcasieu River AOC.

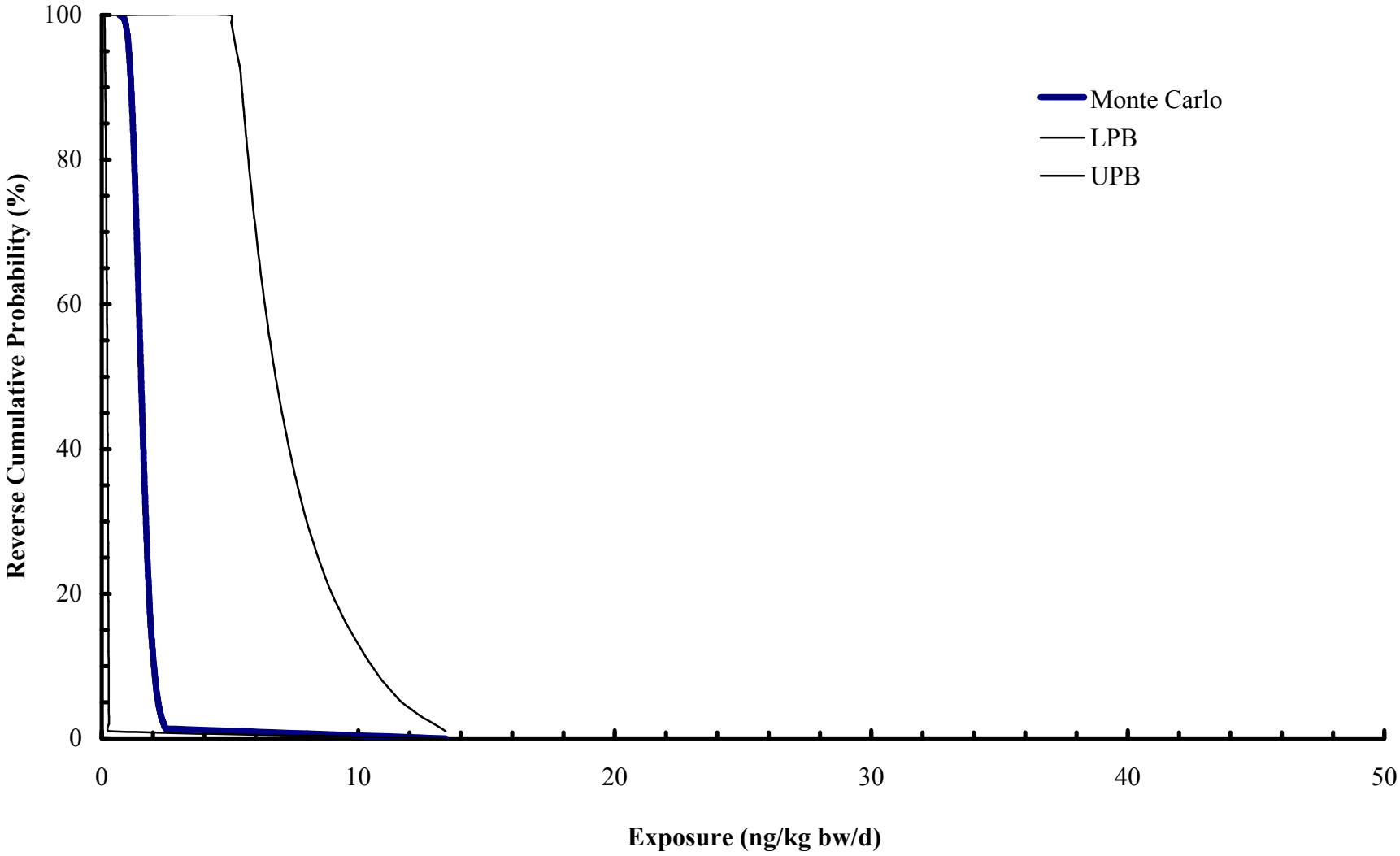

Figure I1-16. Exposure of hypothetical average-sized omnivorous mammals to TCDD and equivalents in the Calcasieu reference areas.

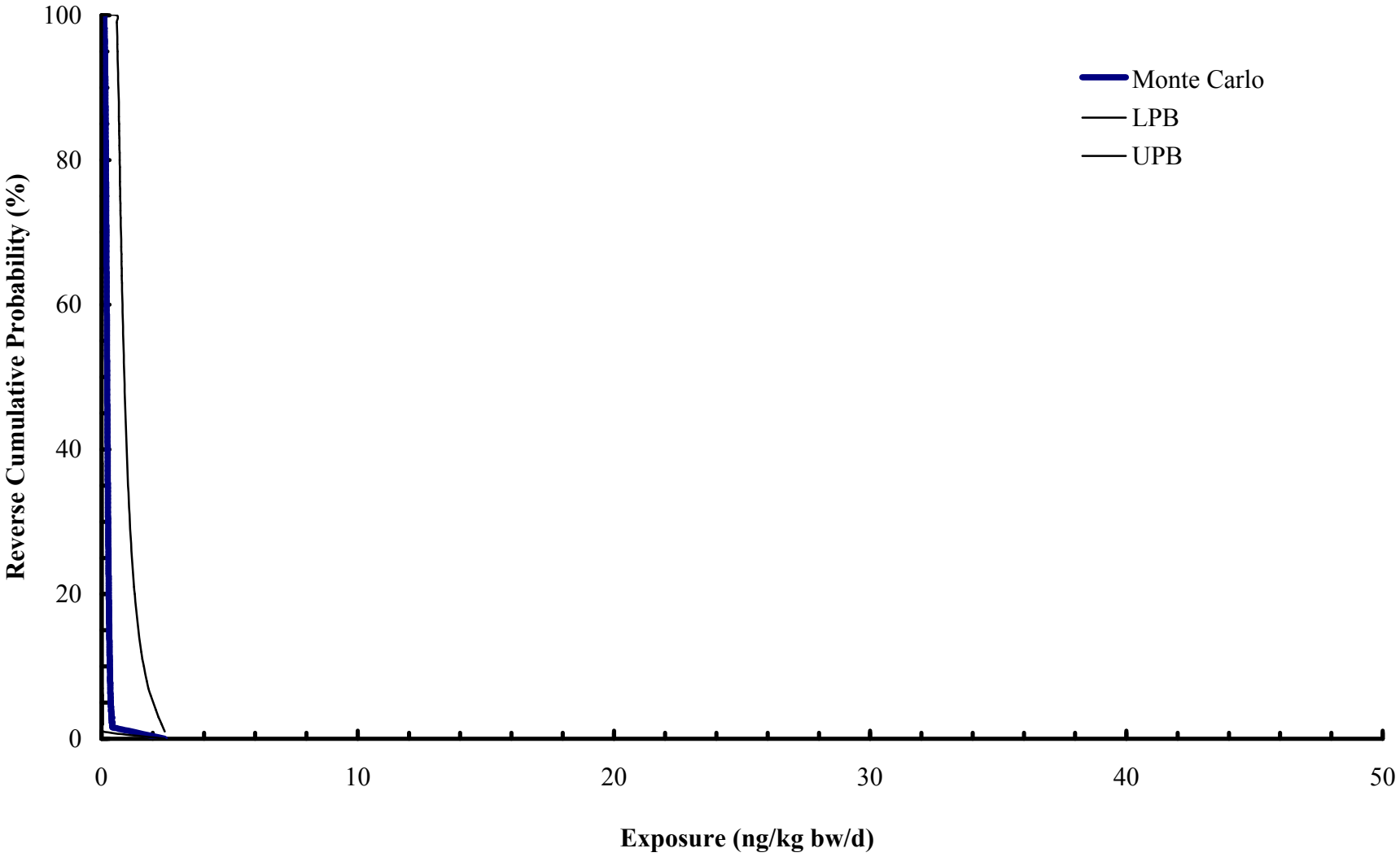

Figure I1-17. Exposure of hypothetical small omnivorous mammals to TCDD and equivalents in the Calcasieu reference areas.

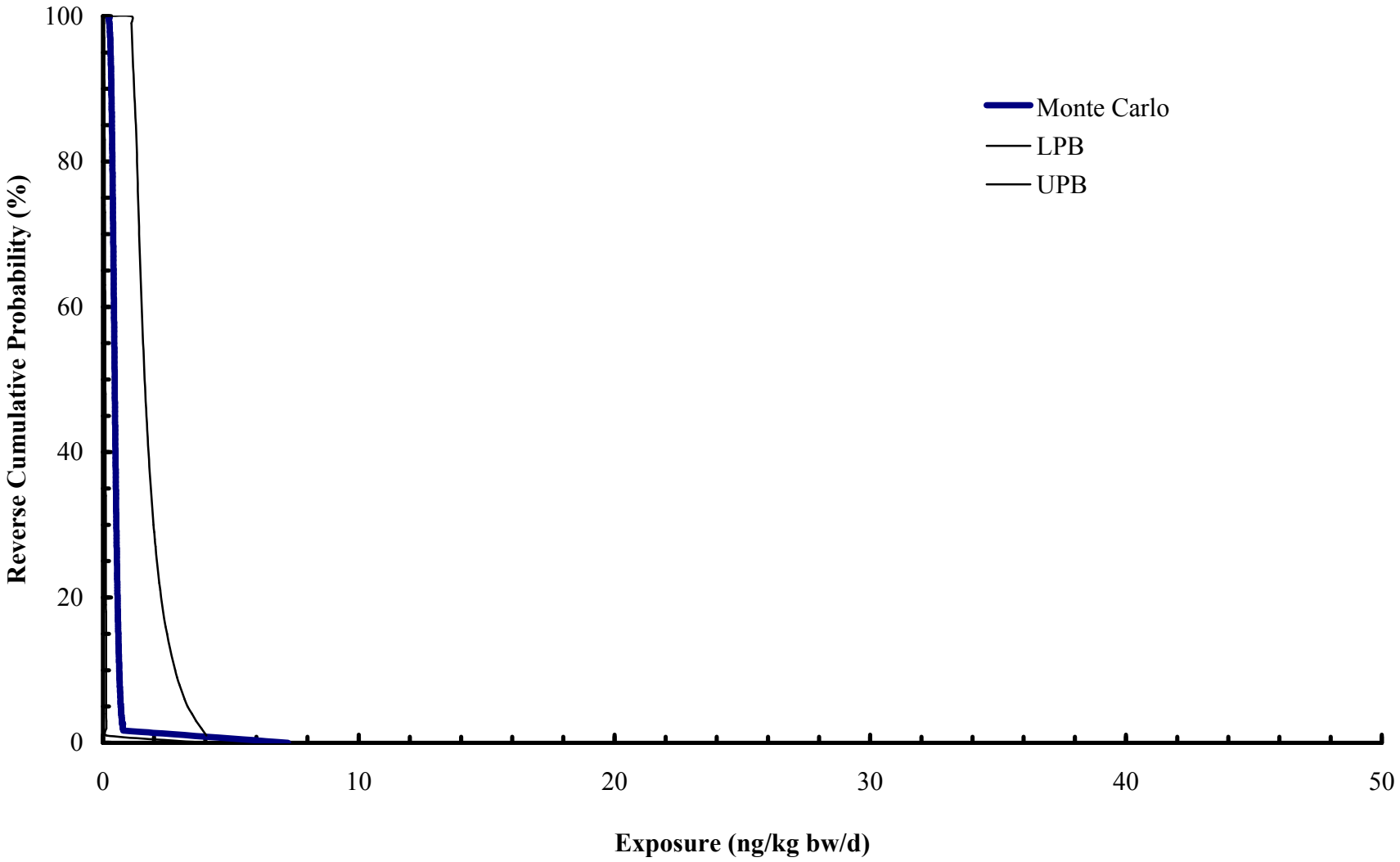

Figure I1-18. Exposure of hypothetical average-sized omnivorous mammals to selenium in Bayou d'Inde AOC.

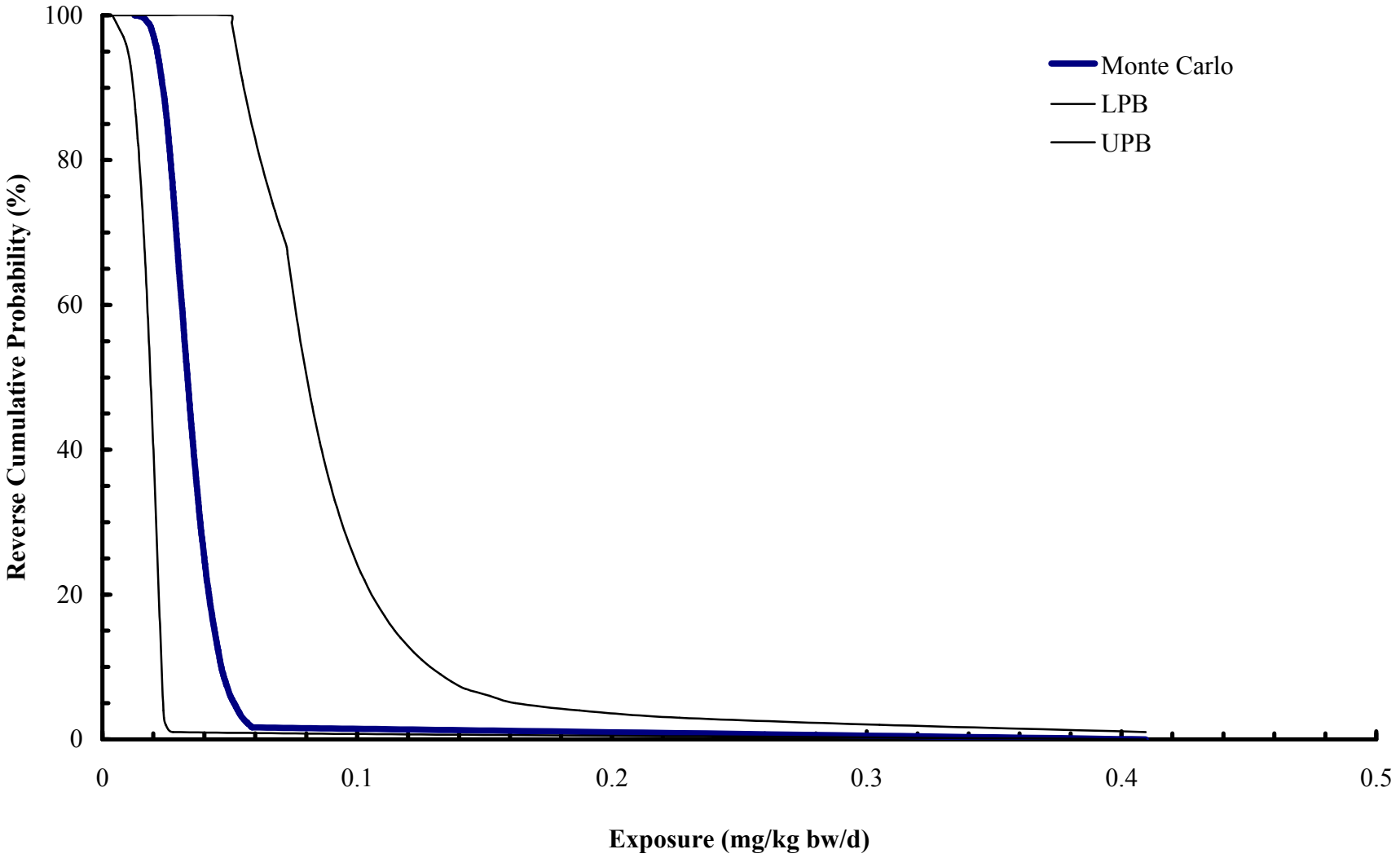

Figure I1-19. Exposure of hypothetical small omnivorous mammals to selenium in Bayou d'Inde AOC.

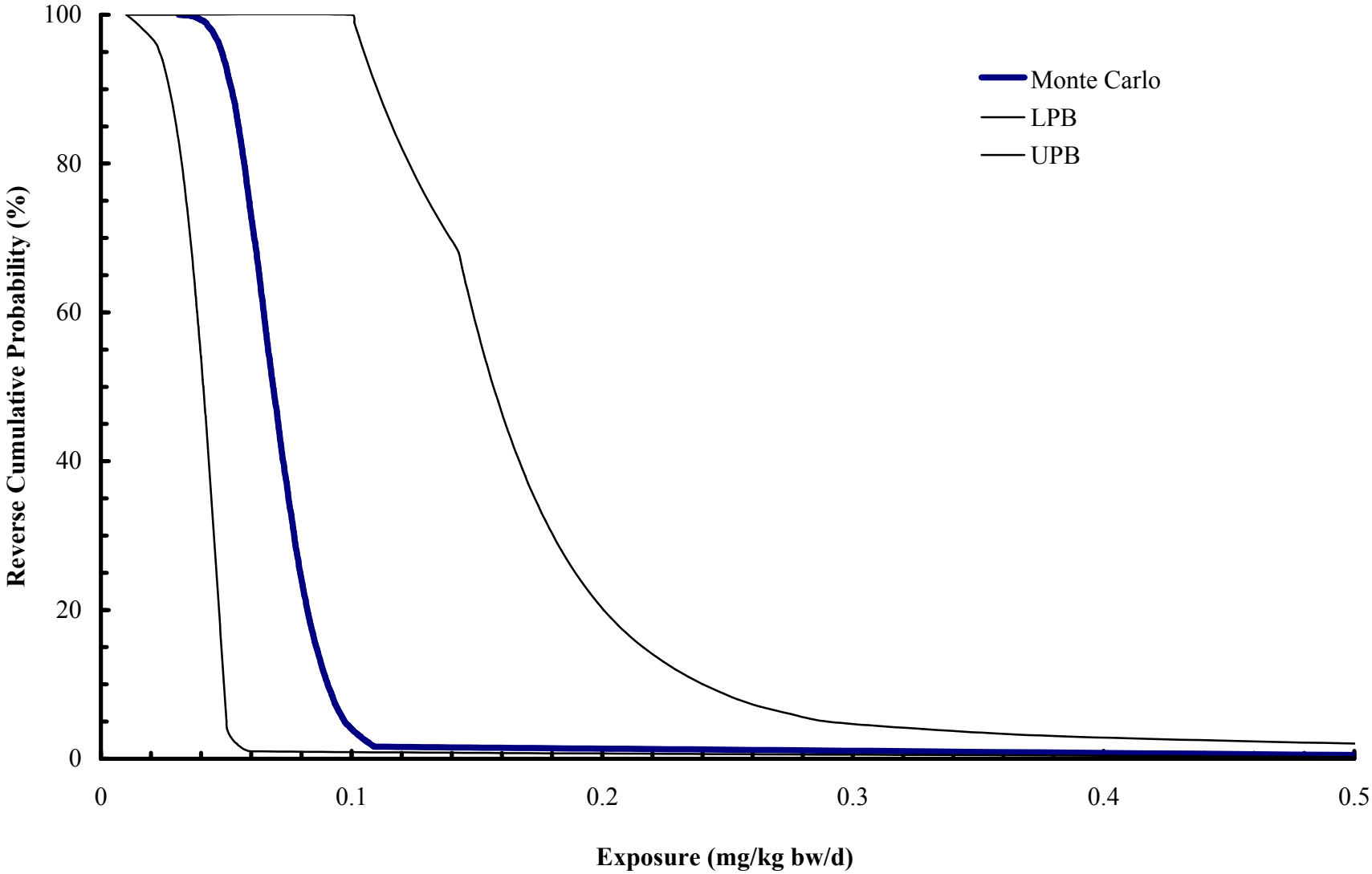

Figure I1-20. Exposure of hypothetical average-sized omnivorous mammals to selenium in Middle Calcasieu River AOC.

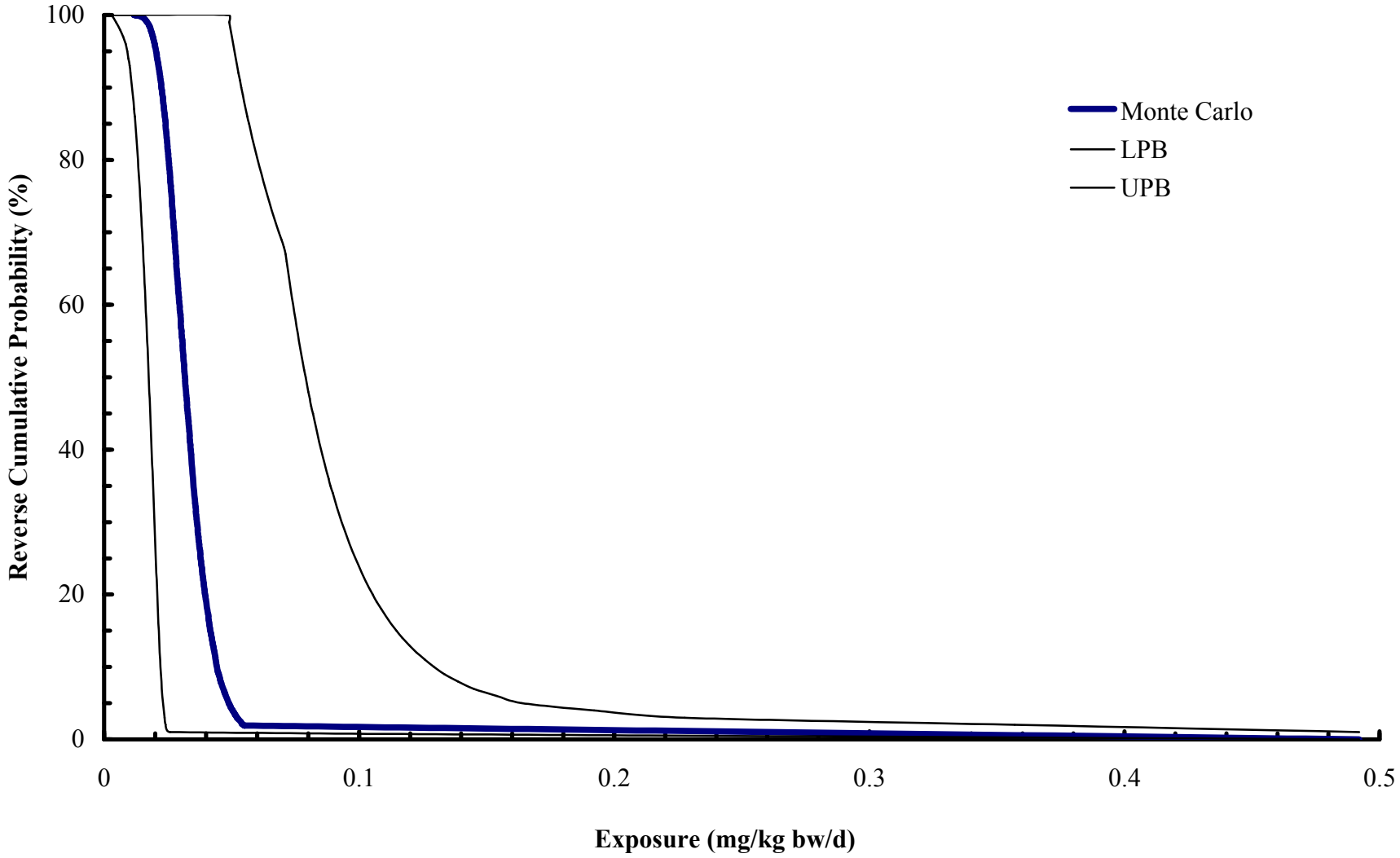

Figure I1-21. Exposure of hypothetical small omnivorous mammals to selenium in Middle Calcasieu River AOC.

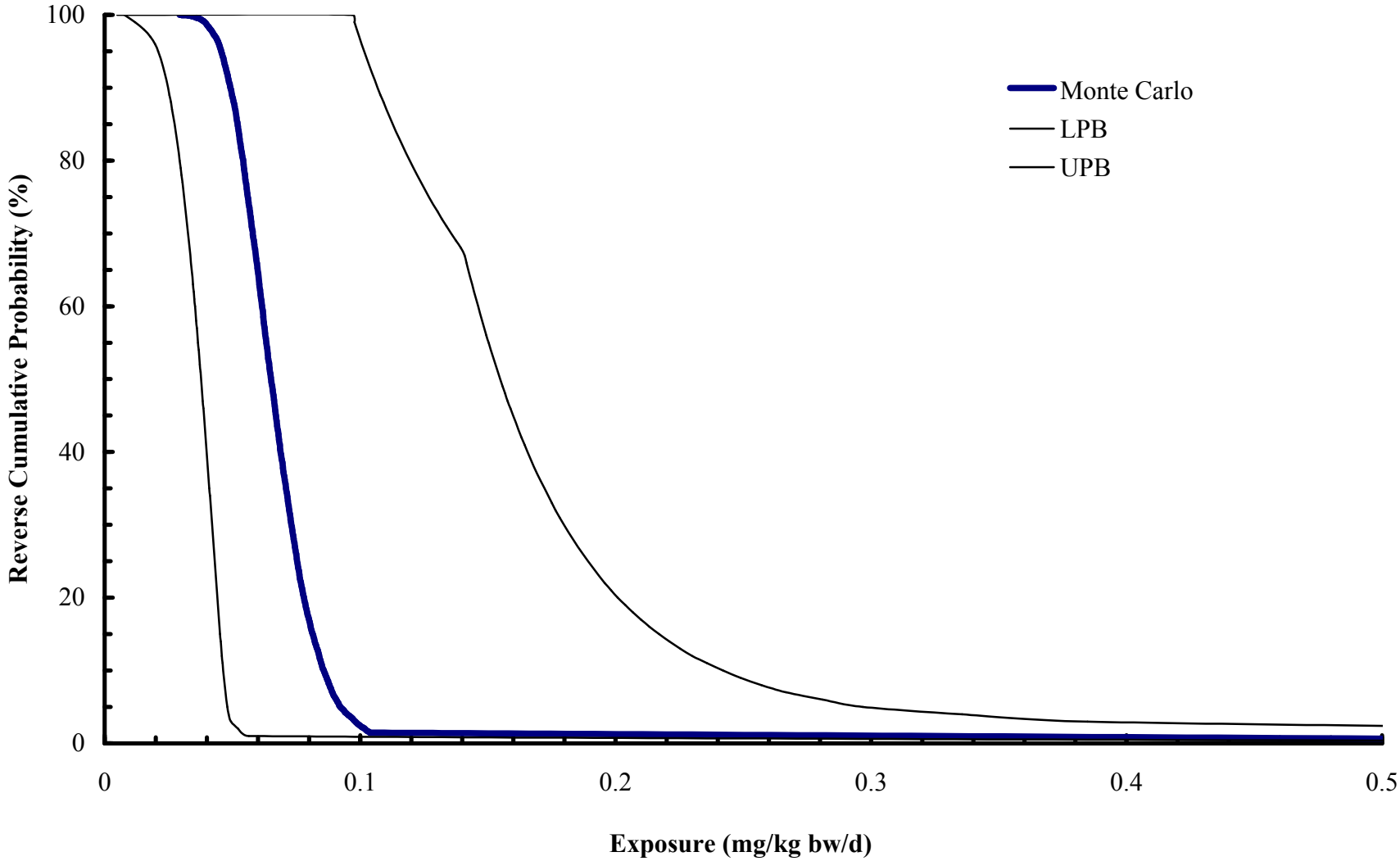

Figure I1-22. Exposure of hypothetical average-sized omnivorous mammals to selenium in Upper Calcasieu River AOC.

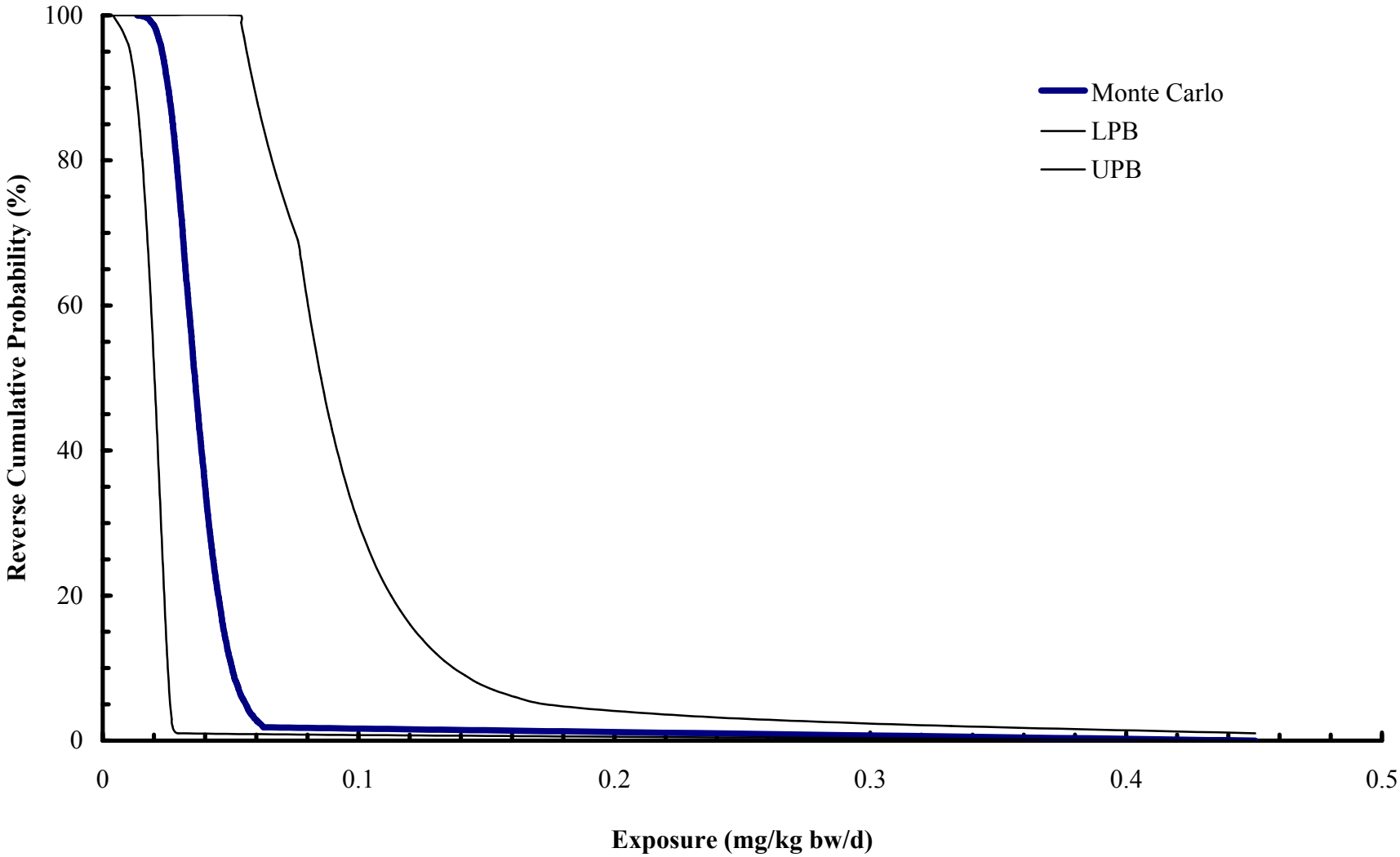

Figure I1-23. Exposure of hypothetical small omnivorous mammals to selenium in Upper Calcasieu River AOC.

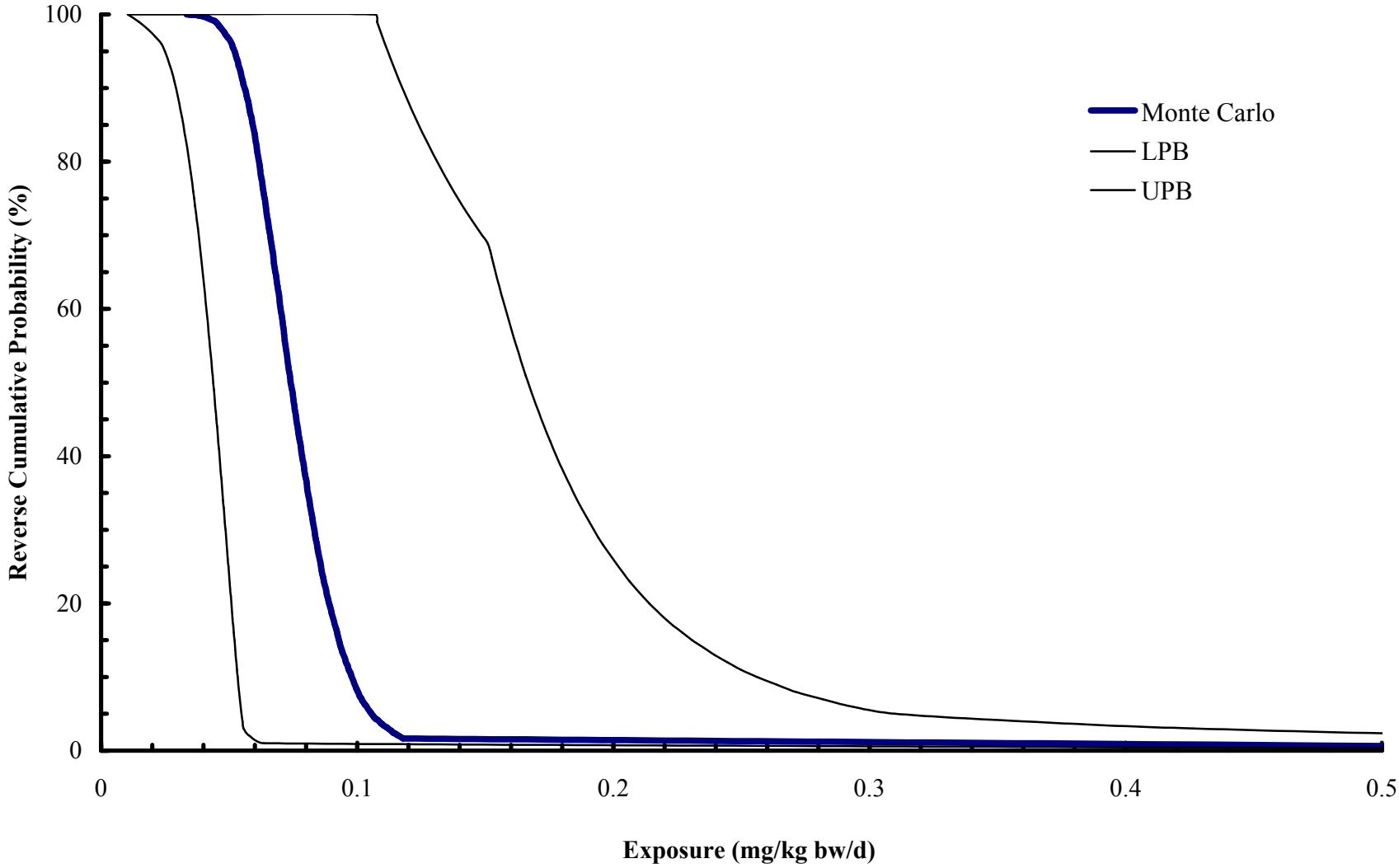

Figure I1-24. Exposure of hypothetical average-sized omnivorous mammals to selenium in the Calcasieu reference areas.

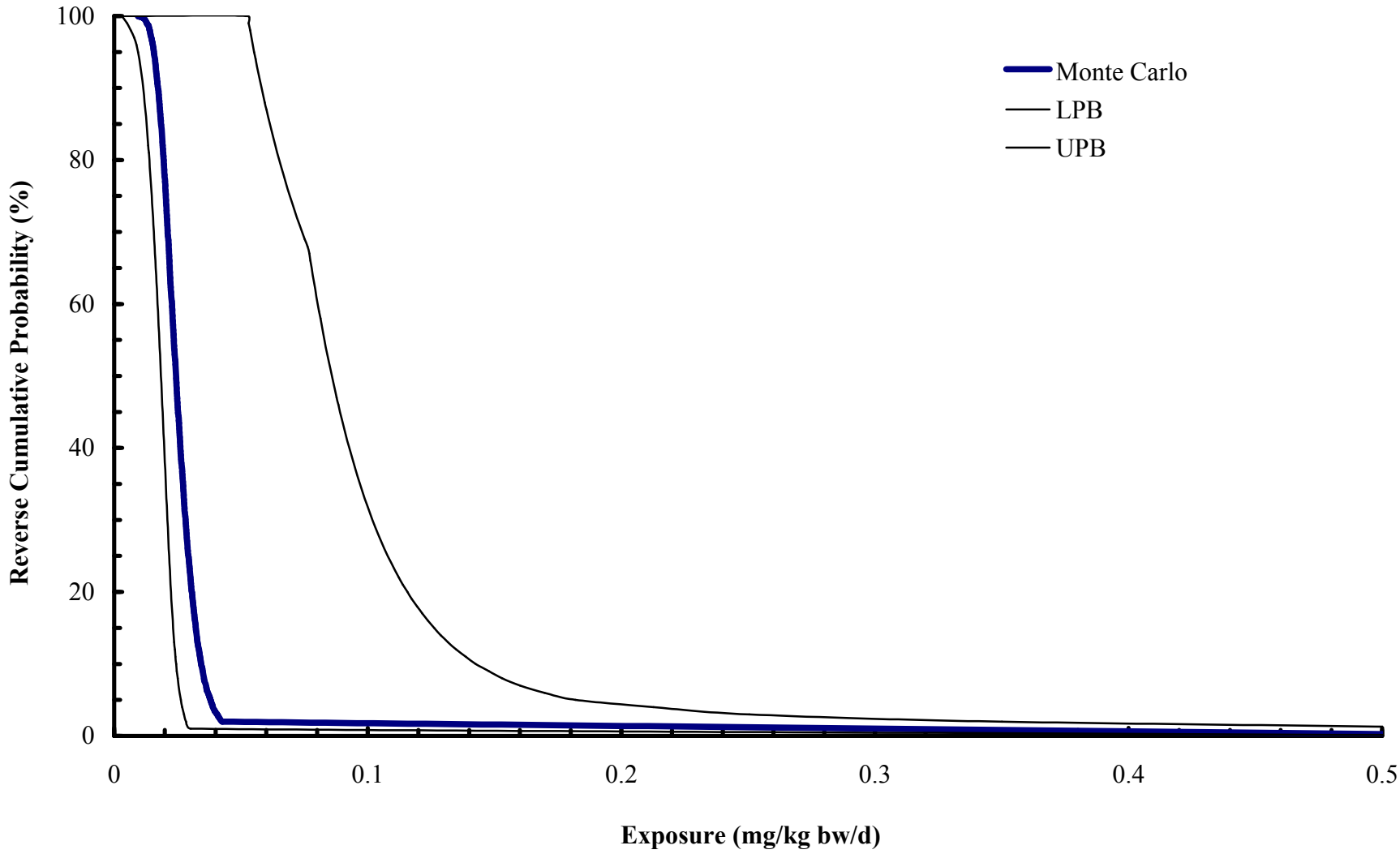

Figure I1-25. Exposure of hypothetical small omnivorous mammals to selenium in the Calcasieu reference areas.

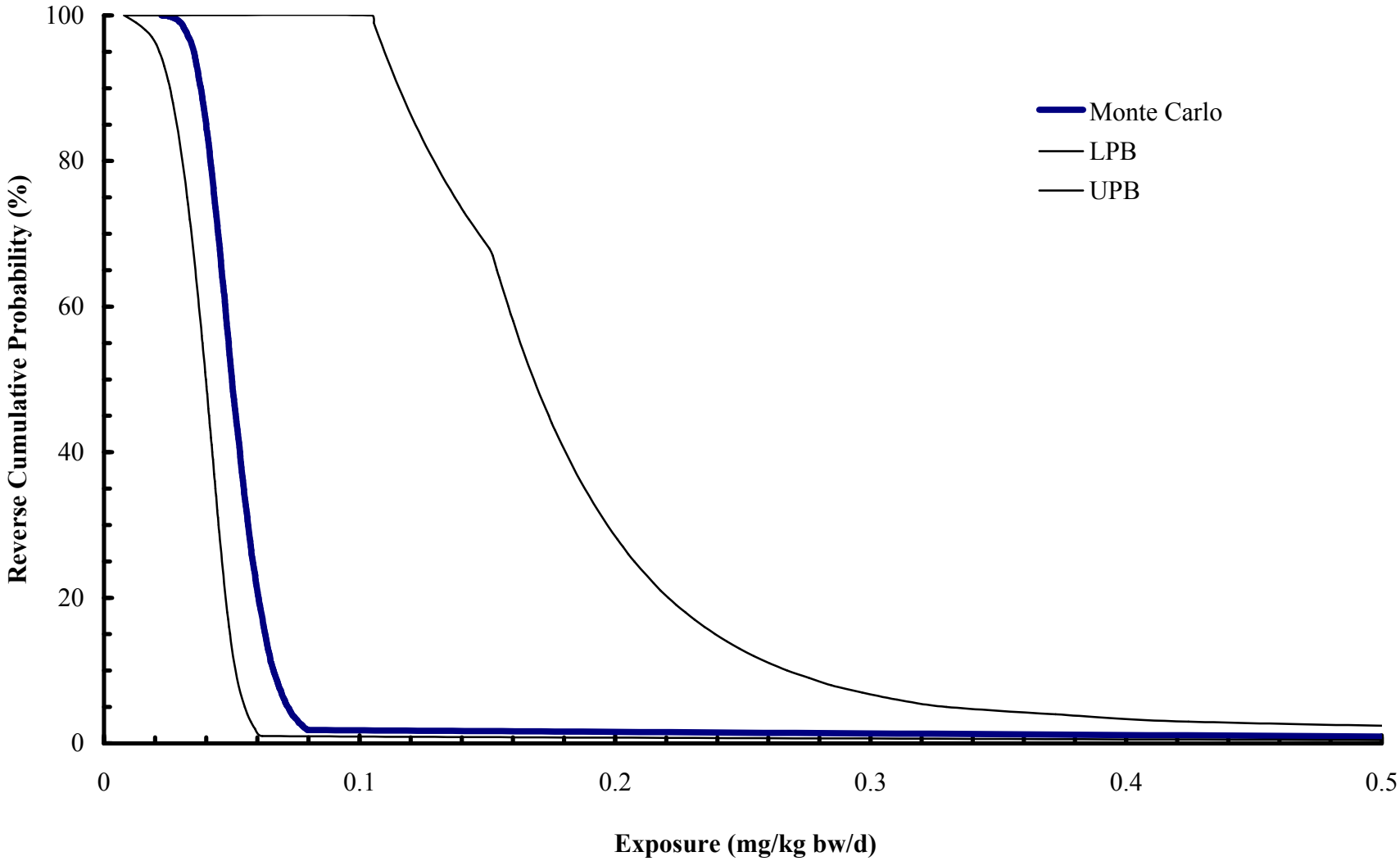

Figure I1-26. Exposure of hypothetical average-sized omnivorous mammals to total PCBs in Bayou d'Inde AOC.

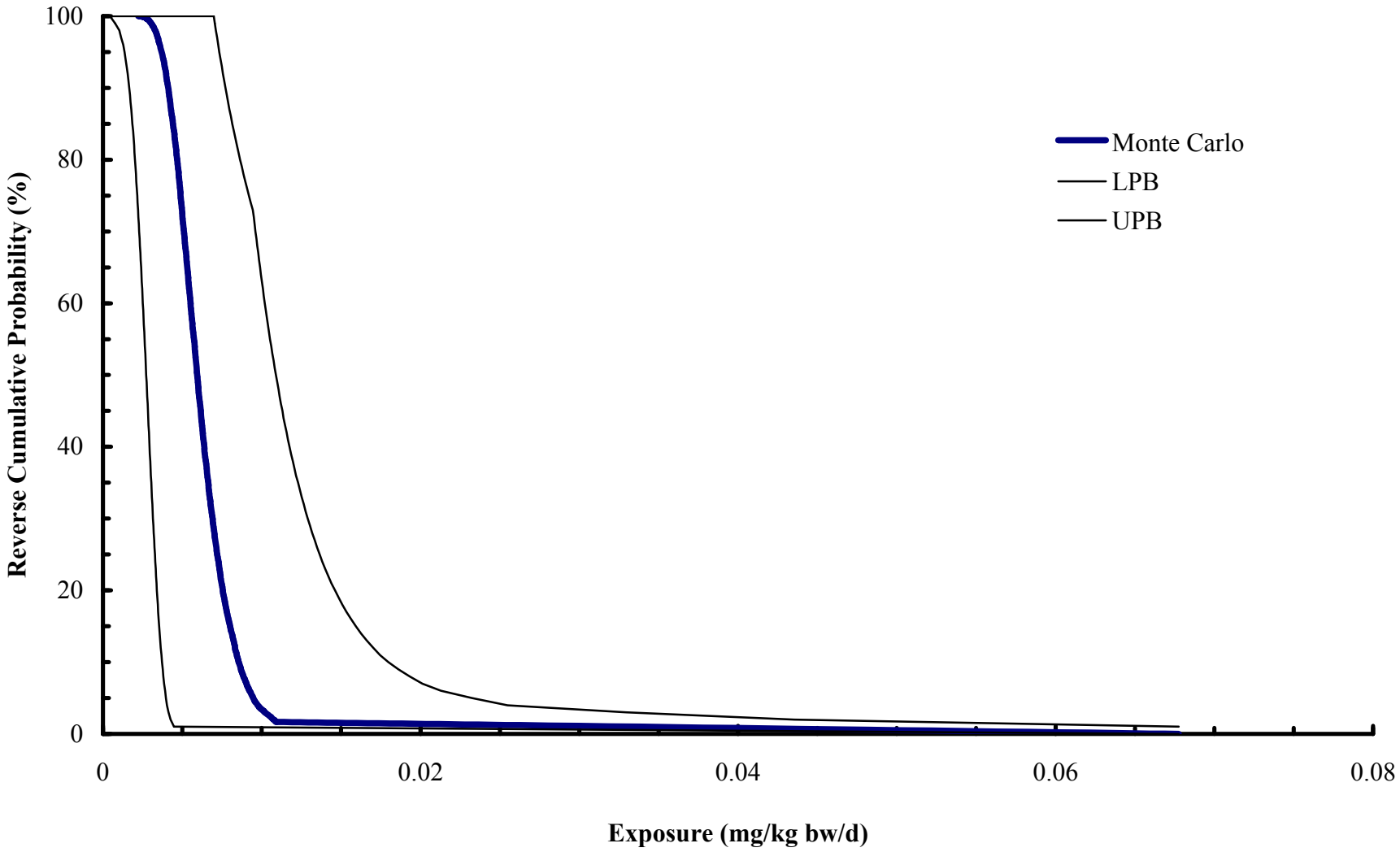

Figure I1-27. Exposure of hypothetical small omnivorous mammals to total PCBs in Bayou d'Inde AOC.

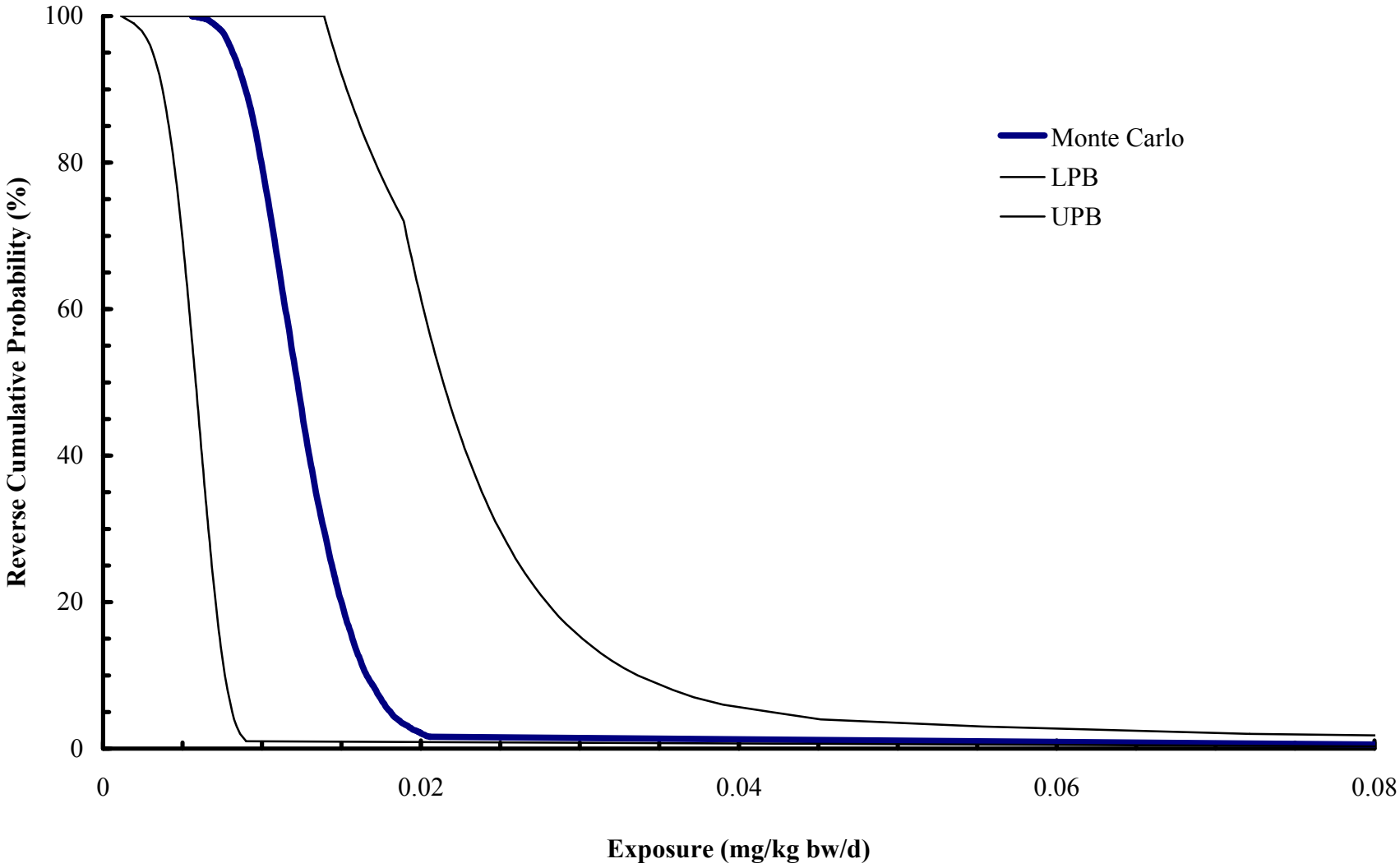

Figure I1-28. Exposure of hypothetical average-sized omnivorous mammals to total PCBs in the Calcasieu reference areas.

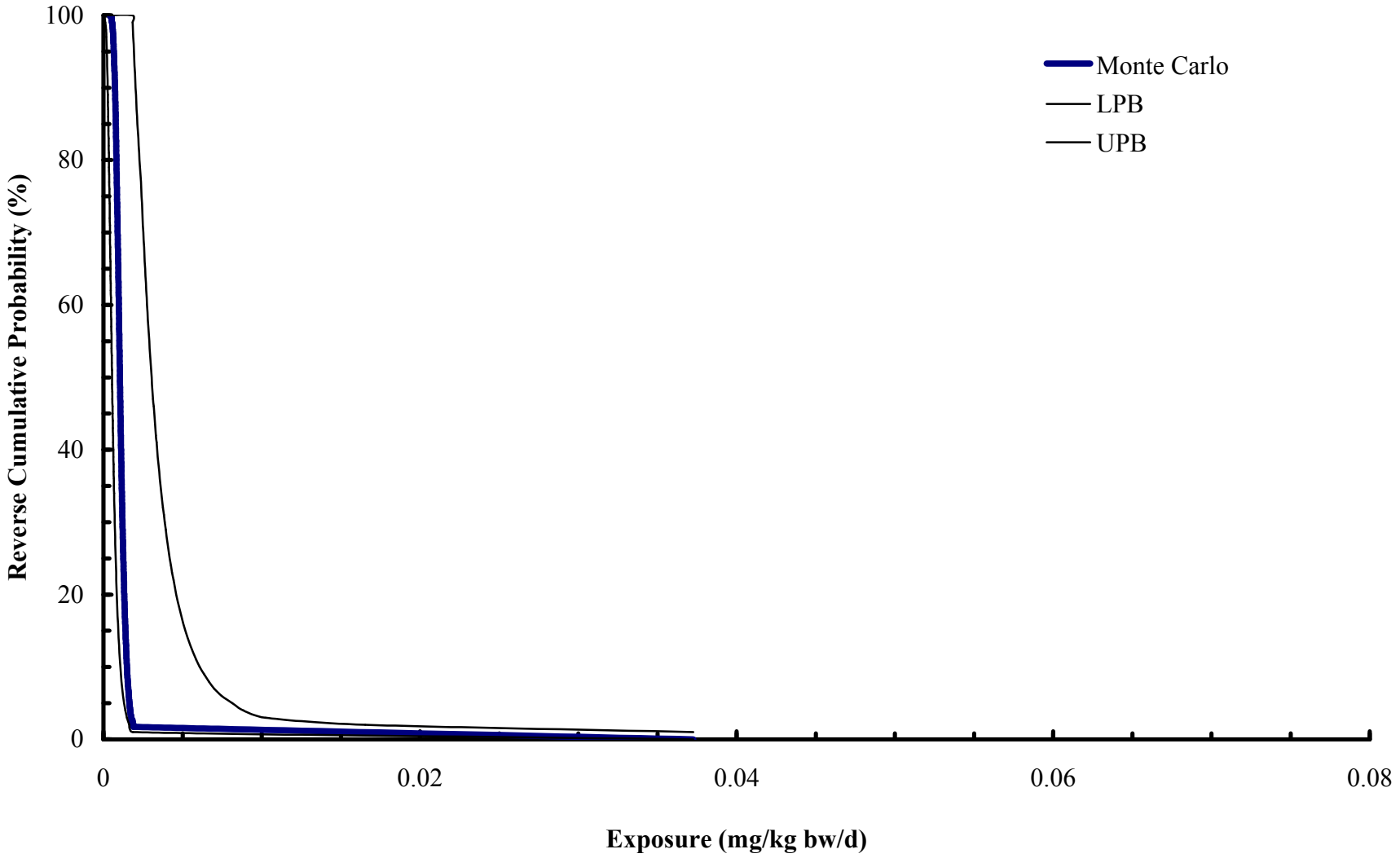

Figure I1-29. Exposure of hypothetical small omnivorous mammals to total PCBs in the Calcasieu reference areas.

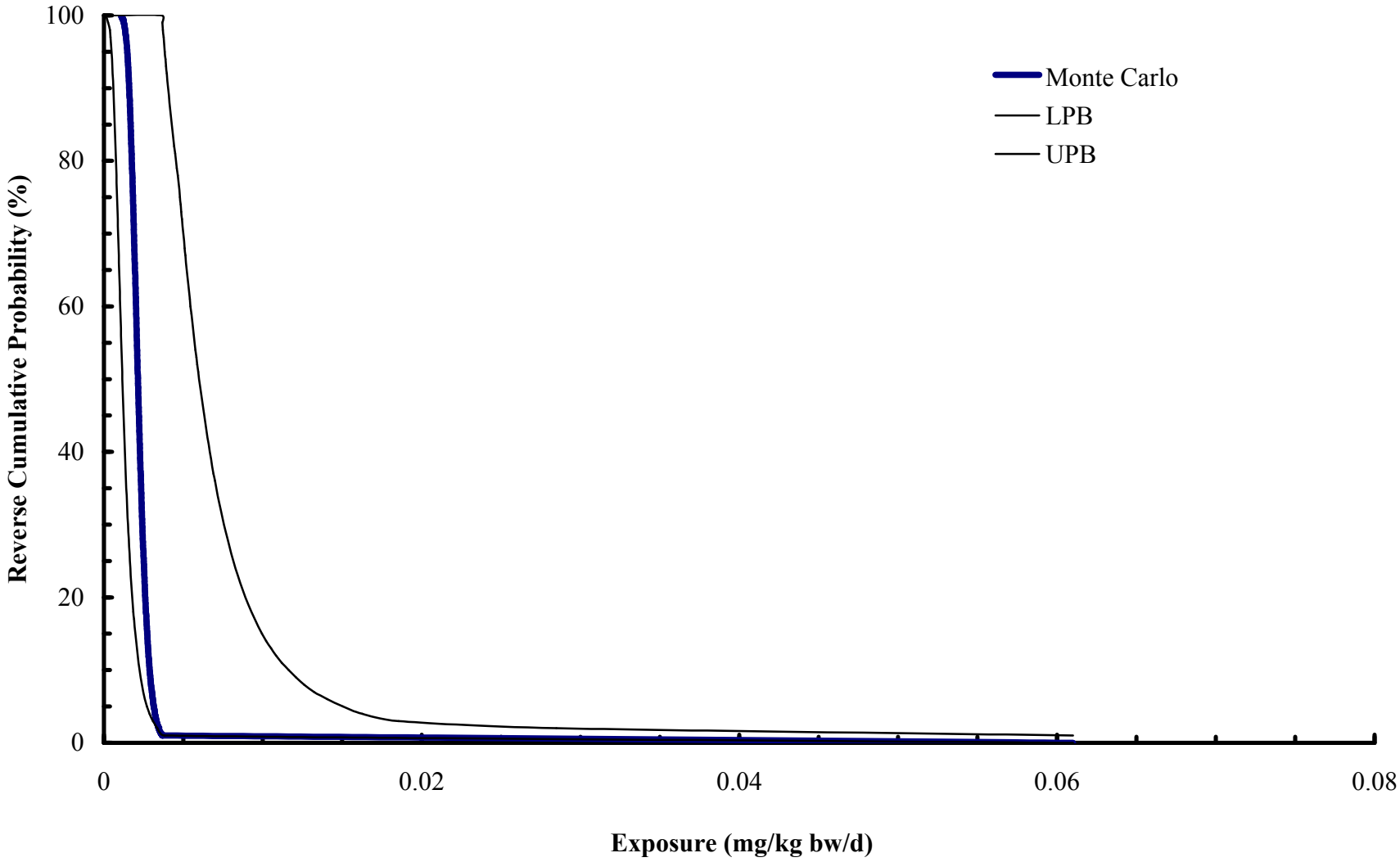

Figure I1-30. Annual geometric mean concentration of Aroclor 1254 in fish fillet from Bayou d'Inde AOC (bars represent minimum and maximum concentrations).

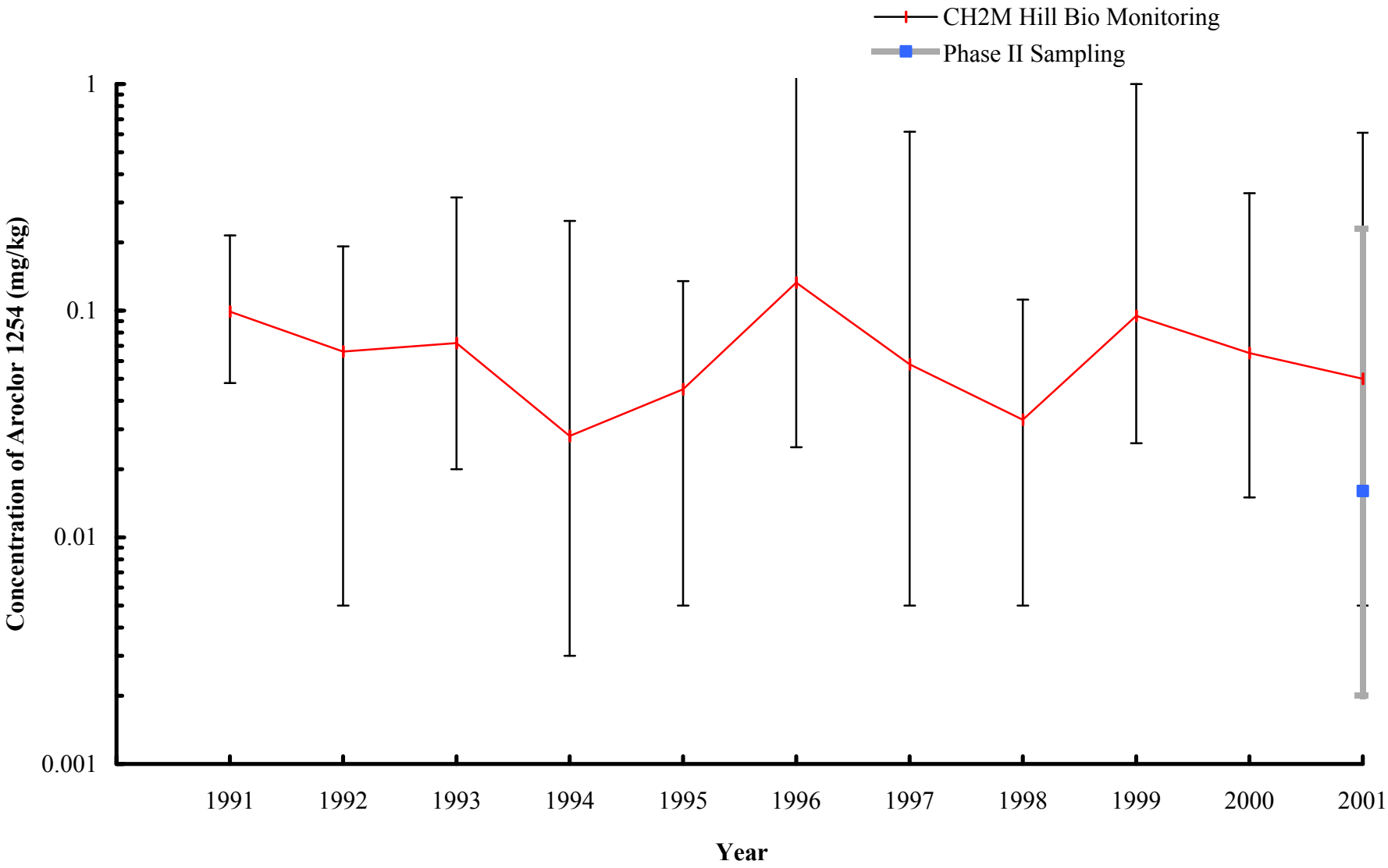

Figure I1-31. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Middle Calcasieu River AOC (bars represent minimum and maximum concentrations).

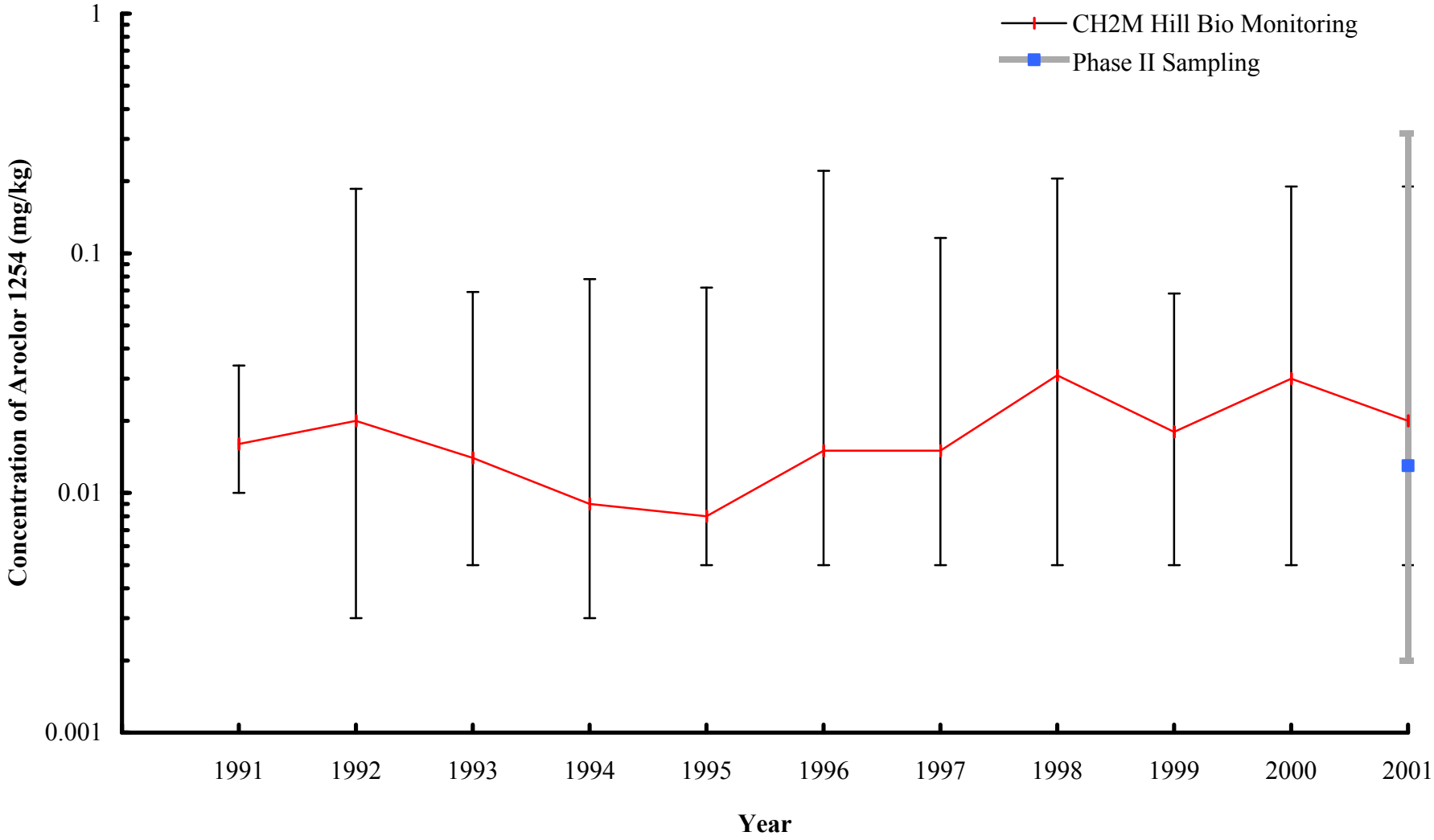

Figure I1-32. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Upper Calcasieu River AOC (bars represent minimum and maximum concentrations).

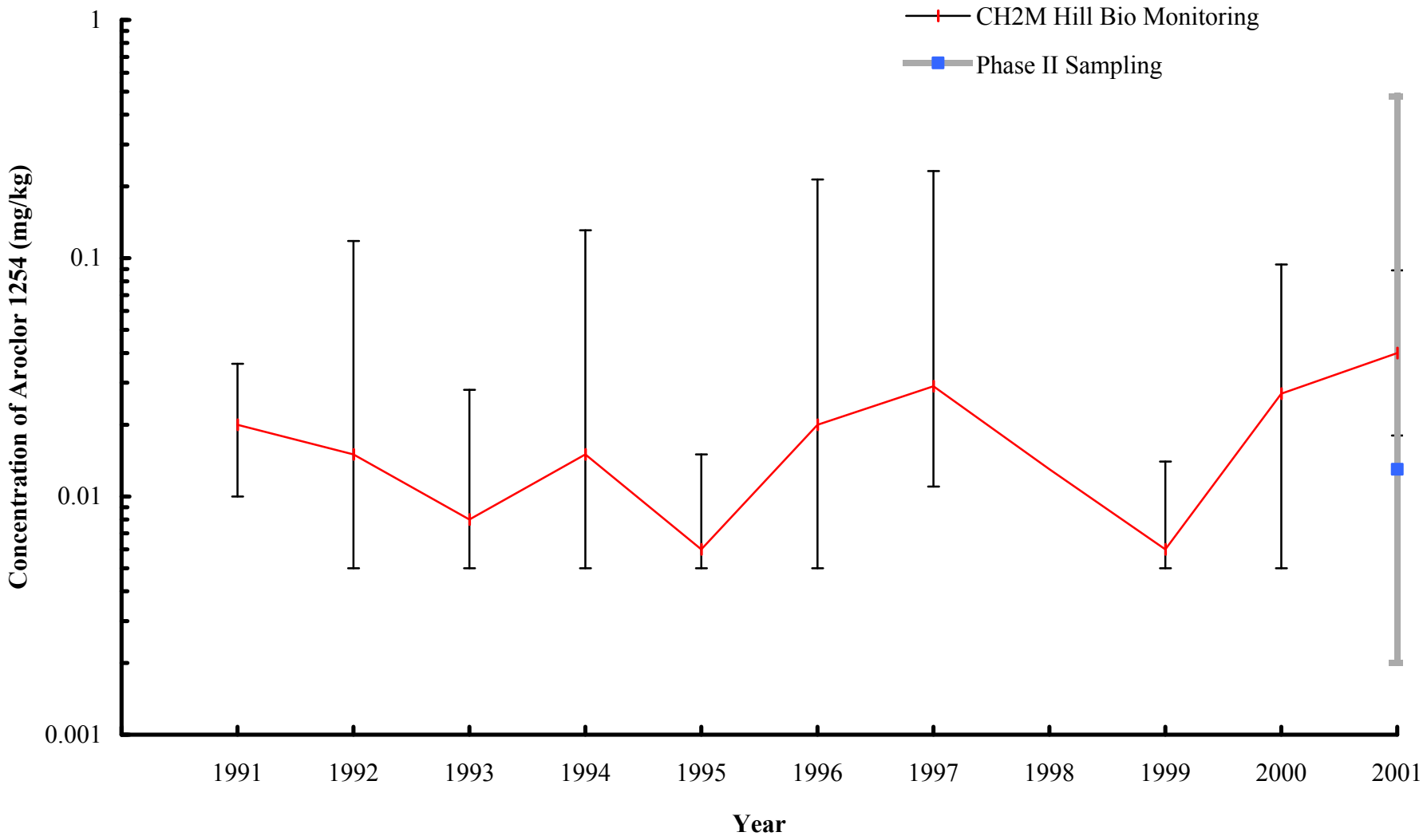

Figure I1-33. Annual geometric mean concentration of Aroclor 1254 in fish fillet from reference areas of the Calcasieu Estuary (bars represent minimum and maximum concentrations).